A CLINICAL STUDY OF MATERNAL AND PERINATAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY

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CERTIFICATE

This is to certify that the dissertation entitled "**A CLINICAL STUDY OF MATERNAL AND PERINATAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY"** is the bonafide original work of **Dr. T. SUREKA** in partial fulfilment of the requirements for **M.D. Branch – II (Obstetrics and Gynaecology)** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in March 2007. The period of study was from May 2004 to March 2007.

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DECLARATION

I, **Dr. T. SUREKA,** solemnly declare that dissertation titled, "**A CLINICAL STUDY OF MATERNAL AND PERINATAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2004-2007 under the guidance and supervision of **Prof. Dr. B. RUPA, M.D., D.G.O.**

The dissertation is submitted to Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree (Branch – II) in Obstetrics and Gynecology.**

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CONTENTS

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KEY TO MASTER CHART

HEADINGS :

O/H : Obstetric History

GA : Gestational Age

U/A : Urine Albumin

BS : Bile Salts

BP : Bile Pigments

BT : Bleeding Time

CT : Clotting Time]

PT : Prothrombin Time

Hb : Haemoglobin

P.Smear: Peripheral Smear

PPH : Postpartum Haemorrhage

SYMPTOMS

1. Nausea, Vomitting
2. Anorexia
3. Fever
4. Abdominal Pain
5. High coloured urine
6. Pruritis
7. Loose stools
8. Clay Stools
9. Itching

SIGNS

1. Jaundice
2. Hepatomegaly
3. Spleenomegaly
4. Ascities
5. Altered sensorium

PREGNANCY OUTCOME

PT : Preterm

LN : Labour Natural

ND : Neonatal death

IUD : Intrauterine death

DIC : Disseminated Intravascular coagulation.

B.term : Borderline term

COMPLICATION

1. Anaemia
2. PIH
3. Oliguria
4. Chronic Active Hepatitis with PHT – Devascularisation done
5. Cirrhosis with PHT
6. Gillbert’s disease

INTRODUCTION

Pregnancy occurring in patients with Jaundice is an important medical and obstetric problem producing adverse effects to both mother and fetus. It often leads to a high maternal mortality and fetal loss. The problem is world wide but in the prevailing conditions of poor nutritional status, anaemia, low hygienic conditions and inadequate treatment facilities in developing countries it assumes a major importance.

Jaundice in pregnancy is an important medical disorder seen more often in the developing countries than in the developed. It is responsible for about 10 percent of Maternal death's in India **(Jeyaram K, 1988, Rao.K.B. 1955).** Because of its frequency, viral hepatitis commands priority recognition as the most important cause of Jaundice in pregnancy. Although there are many reports in the medical literature stating that pregnancy does not exert any adverse effect on the course of infective hepatitis, this is always one of the commonest cause of perinatal mortality, morbidity, maternal mortality, morbidity.

Recent advance in the laboratory techniques to study the serological viral markers, Identification of leptospirosis, modern radiological technique

to study the liver including CT scans, MRI, Ultrasound have facilitated the establishment of the etiology of Jaundice.

In developing countries like India, infective etiology still plays a major role in causing Jaundice. Control of infections causing Jaundice will have an impact on the maternal and fetal health. Risk of perinatal transmission of Hepatitis B Virus to the Neonate can be greatly minimized by the use of immunoglobulin and vaccine for immunoprophylaxis.

Jaundice complicating pregnancy has been the cause for maternal mortality in 11.76% cases during a 3 year study in **Govt. R.S.R.M. Lying in Hospital,** Royapuram, Chennai. Therefore this study in undertaken with an aim to analyse the etiology, course of the disease and maternal and perinatal outcome of jaundiced pregnant women. It is sincerely hoped that this study will help in improving the maternal and perinatal outcome in jaundice complicating pregnancy.

REVIEW OF LITERATURE

Incidence

Absolute figures for the incidence of jaundice in pregnancy are difficult to obtain and different in various countries. Sheila Sherlock in her classical work on liver and its disorders, states that the incidence of Jaundice in pregnancy is 1 out of every 1500 pregnancies (0.067%). Incidence in India varies between 0.92% to 1.46%. With the advent of Australian Antigen **(Hepatitis B Surface Antigen)** by Blumberg in 1965, the etiological classification was made easier. The incidence of Hepatitis B infection in pregnancy in India is reported to be 1 in 425 pregnancies (0.245%).

The true incidence in pregnant women in India may be higher because

1) Many anicteral and mildly icteric patients would not attend the hospital.

2) Missed diagnosis is particularly liable to occur in early pregnancy where Nausea, Vomiting and anorexia are usually attributed to hyperemesis gravidarum.

Liver function tests and Pregnancy

	Non-Pregnant	**Change in Pregnancy**
Bilirubin	2-17µmol/L	No change
Enzymes		
Transaminase	7-40 IU/L	No change
Gamma GT	<30 IU//L	"
5-Nucleotidase	2-17IU/L	"
S. Alkaline Phosphatase	30-130IU/L	Rise, Progressive increase after 5th week to term (1.5 x Normal - Placental Component - Skeletal Component
Prothrombin time	8-14sec	No change
Proteins		
Total Proteins	65-80g/L	Fall (by 10g/l by 16-20th wk)
Albumin	35-55g/L	Fall by 10g/L mostly in first trimester
Globulin	30-50g/L	Rise (Progressive Increase to term)
Fibrinogen	2-4g/L	Rise (Progressive Increase to term)
Lipids		
Cholesterol	4-6.5mmol/L	Rise (Progressive Increase to term)

Triglyceride	<1.5mmol/L	Rise

Physiological, Biochemical and anatomical changes in liver during pregnancy

In normal pregnancy, with adequate nutrition, the metabolic changes seem to be without any effect on liver metabolism and function. In the non pregnant individual hepatic blood flow represents 25-35%of cardiac output. In pregnancy, the increased blood volume is partly redistributed into the splanchnic circulation and great veins. So the portal venous pressure of the gravid uterus on the vena cava results in diversion of a portion of venous return through the azygos system. Venous pressure increases in the esophagus leading to transient engorgement of esophageal veins (varices) in approximately 60% of healthy pregnant women.

Serum levels of alkaline phosphatase, transaminase and LDH rise during labour and delivery. The alkaline phosphatase may remain elevated upto 6 weeks postpartum. The alkaline phosphatase is elevated because of the contribution from placenta. Due to delayed excretion of billirubin, upto 15% of normal pregnant women may have billirubin level of over 1.0mg% and this may account for the increased incidence of pruritis during pregnancy.

In the III trimester, the liver occupies a more posterior superior position. A palpable liver is abnormal in pregnancy and usually indicates underlying liver disease. Palmar erythema and spider naevi are common in normal pregnancy.

Concepts of Jaundice

Jaundice is the yellow discolouration of the skin, sclera and mucous membranes due to increased level of serum Billirubin **(more than 2.5mg/dl)**. Internal tissues and body fluids are coloured yellow, except the brain, as billirubin does not cross the blood brain barrier, except in the immediate neonatal period.

Classification of Jaundice

Based on the etiology, Jaundice in pregnancy can be classified as.

I. Jaundice peculiar to pregnancy.

a) Toxemia of Pregnancy

b) Intrahepatic cholestasis of pregnancy.

c) Acute fatty liver of pregnancy

II. Intercurrent Jaundice in pregnancy

a) Viral Hepatitis

b) Other infections

III. Pregnancy on pre existing liver disease.

a) Cirrhosis and portal hypertension

b) Familial non hemolytic Jaundice

IV. Miscellaneous

Jaundice due to Hemolysis, hepatotoxic drugs, Gall bladder disease.

Ia – Severe preeclampsia – Eclampsia

Liver involvement in preeclampsia – Eclampsia is frequently accompanied by other organ involvement; commonly reffered to as HELLP – Syndrome **(Hemolysis, elevated liver enzymes and low platelets)**

Incidence:

It constitutes 0.1 – 0.6% of all pregnancies and 4 – 12% of patients with pre eclampsia. 69% of women present in the antepartum period **(89% after 27 weeks and 11% before 27 weeks)** and 31% in postpartum within 48 hours**.**

Signs and Symptoms:

Typically right upper Quadrant pain occurs in 65 to 90%, nausea and vomiting in 36 – 50%, Head ache in 31%, Jaundice in 5%. Other signs include weight gain with edema. Hypertension may be absent in 20%.

Criteria for diagnosis:

A) Haemolysis –

1. Abnormal peripheral smear.
2. Increased serum Billirubin > 1.2mg/dl.

B) Elevated liver enzymes.

Increased SGOT > 60IU/L

Increased LDH > 600IU/L

C) Low platelets (DeBoer et al., 1991)

Class I	<50,000 cells/cu.mm
Class II	50,000 to 1,00,000/cu.mm
Class III	1,00,000 to 1,50,000/cu.mm

Classification of HELLP (Weinstein et al., 1982)

Partial	:	One (or) Two features

Complete : All three features

Maternal and Fetal Outcome:

The most important cause of maternal morbidity and mortality is the Development of DIC. Eclampsia, Pulmonary edema, acute renal failure and abruptio placenta can occur. Maternal mortality ranges from 0% to 24%. More than 1/3 of fetuses are born premature or exhibit IUGR. Prematurity increases the risk of perinatal mortality which ranges from 30-60% in different series. Prompt delivery is the effective treatment. Risk of recurrence is 3.4%.

b. INTRA HEPATIC CHOLESTASIS OF PREGNANCY (ICP)

ICP is a benign cholestatic disorder that occurs in second and third trimester of pregnancy and disappears shortly after delivery. The incidence of ICP varies geographically with rates as low as 1-2 per 10,000 pregnancies in India, North America and Australia and as high as 2% of Births in Sweden and 14% in chile.

Etiology:

Genetic predisposition: High incidence has been reported in specific ethnic groups in chile & Sweden. The risk of ICP was increased in women whose mothers or sisters had experienced this disorder. Another

theory is a combined defect in sulfation of estrogens and progestogens together with a defect in canalicular excretion of these metabolites. Patients with ICP may have abnormal progesterone metabolism. So progesterone should not be given in pregnant women with history of ICP **(Reyes H et al 2000)**

Clinical features:

Pruritis occurs in 100% and Jaundice in 10-15% of Patients. The problem typically occurs in III trimester, though 25% of cases presented in II trimester. Serum Billirubin is usually <5mg/dl. Transaminase is 5 times normal, in 40% it is more than 10 time normal. Serum Alkaline phoshatase rises to 2 times the normal. Serum Bile acids are elevated. Measurement of fasting serum total bile acid concentration is a valuable diagnostic test, particularly when a pregnant women experience pruritis with normal aminotransferase levels.

Fetal outcome :

ICP has been associated with poor fetal outcomes including increased incidence of meconium stained amniotic fluid (25%), preterm

labour(20%), fetal distress (22-33%) and still birth (1-2%) **(Rioseco AJ, 1994)**.

Maternal Outcome :

The risk of postpartum hemorrhage is 10-22% as a result of Vitamin K malabsorption. Maternal prognosis is good. Symptoms resolve within 2 days of delivery, enzyme levels normalize within 4-6 weeks; Recurrence rate in subsequent pregnancy is 60-70%.

MANAGEMENT

Medical :

1) Ursodeoxycholic acid (UDCA) is the drug of choice. 14mg/kg/d of UDCA improved maternal pruritis and fetal outcome. UDCA also improves maternal bile acid concentration profile, improves progesterone metabolism and markedly decreases the serum levels of sulfated steroids. UDCA is not teratogenic **(Serrano MA, et al 1998)**.

2) Other drugs in use are Antihistamines, benzodiazepines, low dose Phenobarbital, dexamethasone and S-adenosyl methionine (SAM).

Obstetric :

Fetal outcome is improved by early diagnosis, and aggressive management including intense fetal surveillance, delivery after lung

maturity and administration of drugs that decrease bile salts. **Rioseco et al** proposed that patients without Jaundice should be delivered at 38 weeks of gestation whereas jaundiced patients should be delivered at 36 weeks provided that pulmonary maturity has been achieved.

C. ACUTE FATTY LIVER OF PREGNANCY (AFLP)

Incidence is 1 in 7000 to 14,000 deliveries. It is a rare and potentially fatal disease, complicating a normal pregnancy in the third trimester.

Etiology :

Not known precisely. A genetic component has been suggested although no familial cases have been reported. Recent research suggest that AFLP is associated with mutation in long chain 3-hydroxy acyl COA dehydrogenase (LCHAD), a fatty acid oxidation enzyme. Liver disease in pregnancy occurs most often when the fetal deficiency of enzymatic activity is severe.

Clinical Features:

AFLP occurs usually between 30 and 38 weeks of gestation. The frequency is higher in primipara and in women carrying a male fetus. It

presents with sudden onset of nausea and vomiting in 70%, epigastric pain or right upper quadrant pain in 50-80%. Jaundice usually occur 1-2 weeks after the onset of these non-specific symptoms but pruritis is rare. 50% of patients develop preeclampsia. Serum Billirubin is usually between 5-15mg/dl, serum aminotranferases < 1000 IU/L, prolonged clothing time and leukocytosis. Hypoglycemia and renal dysfunction can occur.

Maternal and Fetal Outcome :

Maternal mortality is very high (50-70%) but recently the rates have come down mainly due to earlier diagnosis and improved intensive care support **(Pereira SP, Williams R, 1997)**. Delivery arrests rapid detoriation of liver function. If not terminated promptly, pregnancy may culminate in fulminant hepatic failure with its attendant complications and a high maternal and fetal mortality. Most patents improve one to four weeks postpartum and recovery is complete.

Treatment :

There is no specific therapy. Early recognition and prompt delivery are recommended to improve maternal and fetal survival. Severely affected

patients should be attended in intensive care units with aggressive treatment of complications.

II (a) VIRAL HEPATITIS :

This is the commonest cause of jaundice in pregnancy. There are 5 distinct types of viral hepatitis – A, B, C, D, E. All but Heptatitis B are RNA viruses. During acute phases, these forms of hepatitis are often clinically similar.

In many cases, infections are subclinical but if clinically apparent symptoms may precede jaundice by 1 to 2 weeks. Symptoms include nausea, vomiting, headache and malaise. Low grade fever is more common with Hepatitis A. When jaundice appears symptoms improve, but the pain and tenderness over liver may persist. Serum aminotransferase levels vary and their peaks do not correspond with disease severity. Peak levels of 400 to 4000 IU/L are usually reached by the time jaundice develops. Serum Billirubin levels usually peak at 5 to 20mg/dl and they typically continue to rise despite falling aminotransferase levels.

Adverse maternal outcome relates to the propensity for developing severe viral hepatitis during pregnancy. Reports on severity differ between developing and developed countries. Most data from India and North Africa reports that epidemics of Hepatitis E are severe among pregnant women. Acute viral hepatitis in an adult in a high prevalence country such

as India is more likely to be due to HEV or HBV rather than HAV. There is widespread exposure to HAV in childhood in our country and hence long term immunity. Maternal mortality in pregnant women with viral hepatitis is 14.3% whereas in non pregnant women it is 5.6%.

Adverse fetal outcome are mainly due to risk of vertical transmission from mother and increased risk of preterm delivery. No excess evidence of congenital abnormalities has been reported.

No specific therapies are recommended for Hepatitis A through E during pregnancy. Interferons, ribavarin, nucleoside analogues, ganciclovir and foscarnet are contraindicated in pregnancy. Only lamivudine is recommended in mothers with chronic active hepatitis B to prevent transmission to the fetus and has been shown safe and effective. There is no indication for termination of pregnancy in viral hepatitis.

HEPATITIS A

It is caused by RNA virus and is transmitted by faeco-oral route. It occurs as epidemics mostly in children rather than adults. Almost universal exposure occurs in infancy and childhood in developing countries like India.

The incubation period is 2-7 weeks. Signs and symptoms are not specific and it may go undiagnosed. Majority are anicteric. Early detection is by identification of IgM antibody which indicates acute infection. During convalescence IgG antibody predominates. It persists and is responsible for lifelong immunity.

Effect of Pregnancy :

Hepatitis A is a self limiting illness noted for its complete recovery. It has the same course during pregnancy as in nonpregnant patient. A chronic carrier state is not recognized. Acute liver failure is rare and especially not common in pregnancy. Risk of vertical transmission is negligent. The risk of preterm birth is increased in acute infection **(Hieber JP et al, 1987)**. Pregnant women exposed to infection should be given prophylaxis with 1ml of immunoglobulin immediately following exposure (CDC, MMWR 1999). It gives a protective efficacy rate of around 80% short term. Neonatal immunoprophylaxis is rarely needed except in preterm as most neonatal infections are mild and life long immunity follows recovery.

HEPATITIS – B :

The agent is a DNA hepadna virus. The incubation period is 3-6 months. The route of transmission by infected blood or blood products. Other infective secretions are saliva, vaginal secretions and semen. It is therefore a sexually transmitted disease.

The first virological marker to appear is HBsAg. HBsAg levels decline after the onset of illness and usually undetectable three months after exposure; if it persists beyond three months it indicates chronic disease. Although HBeAg is invariably present during early acute hepatitis, its persistence indicates chronic infection and active viral replication. Approximately 90% of persons with hepatitis B infection recover completely. Of the 10% who develop chronicity, one fourth develop chronic liver disease which is the most serious consequence of Hepatitis B infection.

Effects on Pregnancy :

Neither the prevalence nor the clinical course is altered by pregnancy. Hepatitis B was seen in 17-20% of pregnant women with viral hepatitis. Over 90% of the women recovered fully. But in those associated with anaemia, malnutrition and diabetis the course may be prolonged and severe. Preterm delivery is increased. Transplacental transmission is

common in acute hepatitis and also in chronic seropositivity. Transmission is maximum through blood and vaginal secretions. In first trimester, transmission rate is only 10% whereas in 3rd trimester, it is 80-90% (ACOG, educational Bulletin, 1998). Thus antepartum HBsAg testing is mandatory. HBV vaccine is safe and effective in seronegative mothers.

Vertical transmission correlates well with HBeAg positivity indicating high viral load in mother. Those HBeAg negative and anti HBe antibody positive do not appear to transmit the infection. 95% of perinatal transmission occurs intrapartum. Following delivery most infected infants are asymptomatic. Others develop fulminant hepatitis and nearly 85% become chronic carrier.

Prevention of neonatal infection :

Infants born to infected mothers should receive HBV human hyperimmune globulin (HBIG) at delivery along with first dose of HBV vaccine.

	Dose	**Time**
HBIG	0.5ml	Within 12 hr
Vaccine	5-10mcg IM	Within 12 hr

		(0, 1 and 6 months)

CDC and ACOG recommends hepatitis B serological screening for all prenatal patients. For high risk mothers, who are seronegative, vaccine can be provided during pregnancy – 20mcg IM at 0, 1 and 6 months.

Recently lamivudine, a nucleoside analog has been used with good safety and efficacy in the last 4 weeks of pregnancy to decrease the risk of vertical transmission **(Van Nunen AB, 2000)**.

HEPATITIS D :

It is a defective RNA virus that is very small and relies on HBV for helper functions. The virus always co-infects with hepatitis B. Chronic infection with B and D hepatitis produces more severe disease and approximately 75% of those affected develop cirrhosis. Prevention of HBV via immunoprophylaxis protects against HDV infection.

HEPATITIS C :

It is a RNA virus of flavivirus family. Transmission is similar to hepatitis B. It is more prevalent in IV drug abusers, multiply – transfused individuals, HIV seropositive subjects and those with high risk sexual

behaviour. The incidence of persistent disease is common after hepatitis C infection.

Effect on Pregnancy :

Prevalence of HCV infection in pregnant women is 1-2%. HCV infection does not interfere with normal pregnancy. Perinatal outcome is not adversely affected. Vertical transmission occurs only when there is HIV co-infections and high maternal HCV RNA levels. Vertical transmission rate is 3-6%.

Unlike for HBV, conventional tests for detecting HCV antigens are not available. Instead diagnosis relies on detecting antibodies to HCV (Anti HCV) and HCV RNA using PCR – based molecular techniques.

HEPATITIS E

It is one of the common cause of viral hepatitis in adults. HEV is a water borne RNA virus that is enterically transmitted. The incubation period is 2-9 weeks. HEV hepatitis is generally mild and self limited but commonly causes fulminant hepatic failure during pregnancy.

Effect on Pregnancy :

The infection rate for men is 2.8% non pregnant women 2.1% and in pregnant women 17.3%. The frequency of disease is 8.8% in I trimester, 19.4% in II trimester and 18.6% in III trimester **(Khuroo MS, 1995)**. The

risk of abortion, preterm birth, IUGR and perinatal mortality are increased **(Mayapadhy et al, 1996)**. Preliminary evidence suggests a high incidence of vertical transmission, around 50% **(Singh S, Mohanty A, 2003)**. The incidence of fulminant hepatic failure in pregnancy is 22% and in non-pregnant it is 2.8%. Hepatic failure was exclusively noticed in last trimester.

b) OTHER INFECTIONS

Malaria :

Malarial infection can be endemic or sporadic. The causative organism is plasmodium species. The incubation period varies from 8-25 days. The infection is transmitted by anopheles mosquito. Clinical features are fever and flu like symptoms including malaise, headache and vomiting, which may occur in intervals. Symptoms are less severe in immune patients. Malaria may be associated with anaemia and Jaundice. Jaundice is due to hepatitis and haemolysis. Liver and spleen enlarge and become tender.

Effect on pregnancy :

Pregnant women have an increased incidence of infection and parasitaemia may be great. Severe anaemia in pregnancy is a major obstetric problem in malaria endemic area. Haemolytic anaemia which can cause jaundice may be severe in pregnancy and hepatorenal syndrome is often the cause of death. Malaria may present as puerperal pyrexia following delivery in women with low immunity. Post partum haemorrhage is an important complication in women with malaria. Malaria can infect placenta leading to maternal anaemia, spontaneous abortion, stillbirth, intrauterine death and low birth weight. Intrauterine death is mainly caused by massive infection of the placenta and persistent high fever. Malarial parasites can cross placenta particularly in non-immune mothers leading to congenital malaria.

WHO recommends malarial chemoprophylaxis throughout pregnancy in endemic areas. It is given as weekly prophylaxis with chloroquine in a dose of 300mg/wk.

LEPTOSPIROSIS :

5-10% of patients with leptospirosis will have hepatic and renal involvement. It is transmitted by direct contact with urine, blood or tissue of an infected animal. They enter through abrasions in skin or through

intact mucous membrane. Incubation period is 2-20 days. More than 90% of patients have relatively mild and anicteric form of leptospirosis. Severe leptospirosis may present as jaundice, renal dysfunction and haemorrhagic diathesis referred as Weil's syndrome.

There is marked elevation of serum billirubin and alkaline phosphatase with moderate elevation of aminotransferases in patients with liver involvement. Diagnosis is based on either isolation of the organism or a rise in antibody titre in Microscopic slide agglutination test (MAT). The latter is commonly used.

Effect on pregnancy :

Perinatal mortality is high in leptospirosis. 30% of pregnancies ended in abortion or preterm death **(Shaked Y, Samra D, 1993).** Vertical transmission is rare.

Penicillin G in a dose of 1.5million units IV quid is still the drug of choice. Other alternative are ampicillin and erythromycin. Doxycycline is contraindicated in pregnancy.

Typhoid :

Enteric fever is endemic in most developing regions especially in India. It is transmitted by ingestion of contaminated water or food. Incubation period is 3-21 days. The most prominent symptom is prolonged fever. It is seen in 75% of cases. Gastrointestinal symptoms are quite variable. Only 20-40% of patients have abdominal pain. Hepatic dysfunction, through less common can occur in patients with typhoid fever. Liver function tests will be moderately elevated.

Effects on Pregnancy :

The acute infection in pregnant women run a similar course to those in non pregnant women. Transplacental infection has been recorded**.** Around 3% of patients with severe form of typhoid fever may go for spontaneous abortions and preterm labor. But effective antibiotic treatment has made this outcome less common.

Chloramphenicol still remains the most effective treatment, but many clinicians prefer ciprofloxacin, ceftriaxone or azithromicin.

Herpes Viruses :

All members of herpes group – Herpes simplex, varicella, cytomegalovirus and epstien barr virus can cause hepatitis. More than half

are associated with immunosuppression. Jaundice is not invariable and mucocutaneous stigmata may be absent. Herpes simplex hepatitis carries a grave prognosis. Mortality exceeds 90% even with treatment. Herpes simplex type II is most common in 3rd trimester. Symptoms are three times more common in pregnancy. The virus can cross placenta but it has no relation with congenital malformation. HSV II acquired at birth from cervical and vaginal contamination can lead to disseminated infection in babies with 90% mortality. Pregnancy is a high risk factor and has been associated with a high maternal and fetal mortality rate. Acyclovir appears to improve the outcome.

III) CIRRHOSIS AND PORTAL HYPERTENSION

Pregnancy in a cirrhotic patient is rare because of reduced fertility and older age. In patients with non-cirrhotic portal hypertension (NCPHT), liver function and fertility are relatively well preserved**.**

In pregnant women, post necrotic cirrhosis due to chronic B or C viral hepatitis is the most common cuase **(Williams Obstetrics, 2005)**. Causes for non-cirrhotic portal hypertension are :

1) Extra hepatic portal vein obstruction

2) Non-cirrhotic portal fibrosis

3) Congenital hepatic fibrosis

4) Polycystic liver disease, etc.

Perinatal Outcome :

Fetal loss is high in advanced cirrhosis. Spontaneous abortions, stillbirths, IUGR and neonatal deaths are all increased **(Schreyer. P, 1982)**. The perinatal mortality rate in cirrhotic patients is 10-38% **(Russel, MA, Craige SD, 1998)**. But Indian studies reported that patients with NCPHT had nearly same pregnancy outcome as general population except for increased incidence of abortion (20%). Fetal outcome was improved in patients who underwent endoscopic obliteration of varices or decompression shunt surgery prior to conception.

Maternal Outcome :

Pregnancy profoundly affects systemic haemodynamics and may contribute to rapid detoriation of portal hypertension including increased risk of variceal haemorrhage. These haemodynamic changes are maximum in 2^{nd} trimester and hence the risk of haemorrhage is greatest during this period.

Maternal Complications include :

1) Hepatic failure and hepatic encephalopathy

2) Variceal haemorrhage

3) Post partum haemorrhage

4) Rupture of splenic artery aneurysm

5) Spontaneous bacterial periotonitis.

Women with cirrhotic portal hypertension develop maternal complication more frequently than women with NCPHT. Post partum haemorrhage occurs in 7-26% of patients and is more common in cirrhotic patients. Jaundice and hepatic encephalopathy are often precipitated by haemorrhage and hypotension.

Maternal mortality is 4-7% in NCPHT whereas it is 10-18% in patients with cirrhosis. **(Sriram PV, Goenka, MK, Singh K, 1999)**. Portal Decompression before pregnancy decreases mortality.

Congenital non-haemolytic Hyperbilirubinemia : GILBERT'S DISEASE:

Gilberts disease presents as mild fluctuating Jaundice which may only be noticed when the patient is tired, dehydrated or calorie depleted. As all of these may occur during normal pregnancy, gilbert's disease may be diagnosed for the first time during pregnancy. Serum billirubin is elevated, but liver enzymes are normal, there is no sign of any systemic disease and

no symptoms other than jaundice. Maternal and perinatal outcome are not affected by the disease. It is relatively common in Western Societies with the prevalence rate of 1-2%. There are no significant risks with the diagnosis but precipitating factors should be minimized or avoided.

AIM OF THE STUDY

1) To study the incidence of jaundice in pregnancy in relation to Age, parity and duration of pregnancy.
2) To study the effect of jaundice in pregnancy in terms of perinatal morbidity and mortality.
3) To study the maternal mortality and morbidity in pregnancies complicated by Jaundice.

STUDY DESIGN

Prospective Cohort Study

MATERIALS AND METHODS

MATERIALS

All cases of pregnancy with jaundice in all the trimesters of pregnancy admitted to the Govt. RSRM lying-in Hospital, Chennai during the period of 2004 to 2006 were taken up for study.

METHODS

The cases were studied as per the proforma appended. Demographic features such as Age, Parity, socioeconomic status, previous obstetric history, gestational age were documented at admission.

Past history including history of jaundice in the previous pregnancy, history of contact with jaundice, history of injections, history of blood transfusions, history of intravenous drug abuse and family history of jaundice were noted.

In the present history, symptoms like fever, nausea, vomiting, anorexia, diarrhea, colour of urine and motion, pruritis and history of bleeding episodes were asked for. The above data were collected in a structured proforma.

Thorough general and systemic examination was carried out for each patient. In addition evidence for anaemia, hepatomegaly, spleenomegaly ascites and purpuric spots were recorded.

Medical gastroenterologist opinion was sought and management protocol was noted. Investigations included urine for albumin, sugar, bile salts and bile pigments, complete haemogram with platelet count, bleeding time, clotting time, prothrombin time, pheripheral smear for malarial parasites, VDRL, blood widal, liver function tests including serum billirubin, transaminases, alkaline phosphatase, serum proteins, albumin, serum fibrinogen, blood urea and serum creatinine.

All patients were screened for viral markers for hepatitis B and C infections and also for leptospirosis. Likewise the diagnosis of intrahepatic cholestasis of pregnancy and acute fatty liver of pregnancy were done by clinical presentation and biochemical parameters. Liver function test was repeated every week in all patients till the time of recovery and discharge from the hospital. The patients were followed up for pregnancy outcome. Ultrasound abdomen was done in patients with chronic liver disease.

OBSERVATIONS

A prospective study of all antenatal patients admitted with jaundice in pregnancy at the Government R.S.R.M. Lying-in Hospital, Chennai from January 2004 to August 2006 was undertaken.

Total number of antenatal admissions during this period is 40219. Total number of patients who had Jaundice is 51 and these form the present study group.

1) INCIDENCE OF JAUNDICE COMPLICATING PREGNANCY

The incidence of jaundice complicating pregnancy during this period in our hospital is 1.26 per 1000 population. It is given yearwise in Table 1.

TABLE – 1

Year	Total antenatal admissions	No. of Jaundiced Pregnant Women	Percentage	Incidence per 1000 population per year.
2004	14264	19	0.13	1.3
2005	14928	18	0.12	1.2
2006	11027	14	0.12	1.2

2) DISTRIBUTION OF CASES ACCORDING TO ETIOLOGY

Among the total antenatal admissions of 40219, 51 patients developed jaundice. This constitutes the incidence of jaundice complicating pregnancy as 1.26 per 1000 population during this study. Out of 51 patients, 36 cases were due to viral hepatitis. 11 cases were HBsAg positive and 2 were positive for Anti HCV.

TABLE – 2

Sl.No.	Diagnosis	No. of Cases	Percentage	Incidence per 1000 population
1.	Viral Hepatitis	35	68.5	0.9
2.	HELLP Syndrome	4	7.8	0.1
3.	Chronic liver disease and PHT	3	5.8	0.074
4.	Intra hepatic cholestasis of pregnancy	2	3.9	0.049
5.	Malaria	2	3.9	0.049
6.	Leptospirosis	2	3.9	0.049
7.	Typhoid	1	1.9	0.024
8.	Combined malaria	1	1.9	0.024

	and leptospirosis			
9.	Gilbert's Disease	1	1.9	0.024

3) AGE INCIDENCE

The patients in study group were in the age ranging from 19 – 35 years. Nearly 58% of jaundiced patients were between 20-24 years. 30% of patients were between 25-29 years.

TABLE – 3

Sl.No.	Age Group in Years	No. of cases	Percentage
1.	15-19	2	3.9
2.	20-24	30	58.8
3.	25-29	15	29.4
4.	30-35	4	7.8
	Total	51	100

4) INCIDENCE IN RELATION TO GRAVIDITY

Maximum number of cases occurred in primigravida (58.8%). This is shown in Table 4.

TABLE – 4

Sl. No.	Gravidity	No. of cases	Percentage
1.	Primigravida	30	58.8

2.	Second Gravida	12	23.5
3.	Third Gravida	8	15.6
4.	Fourth Gravida	1	1.9
	Total	51	100

5) INCIDENCE IN RELATION TO GESTATIONAL AGE

Majority of patients (76.4%) were in 3rd trimester. 20% were in 2nd trimester. 2 patients developed jaundice post partum.

TABLE – 5

Sl.No.	Period of Gestation	No. of Cases	Percentage
1.	I Trimester	-	-
2.	II Trimester	10	19.6
3.	III Trimester	39	76.4
4.	Post partum	2	3.9
	Total	51	100

6) ANALYSIS OF PRESENTING SYMPTOMS

The number of cases with percentage of various symptoms are shown in Table 6.

TABLE – 6

Sl. No.	Symptoms	No. of Cases	Percentage
1)	History of fever	35	71
2)	Nausea and vomiting	28	54.9
3)	Anorexia	28	54.9

4)	Pruritis	22	43.1
5)	High coloured urine	21	41.1
6)	Upper abdominal pain	16	31.3
7)	Clay stools	2	3.9
8)	Loose stools	2	3.9

7) ANALYSIS OF CLINICAL SIGNS :

The clinical signs were not similar in all patients. Jaundice was present in almost all cases followed by hepatomegaly. The number of cases with percentage of various clinical signs are shown in Table 7.

TABLE – 7

Sl. No.	Clinical Signs	No. of Cases	Percentage
1.	Jaundice	49	96
2.	Hepatomegaly	8	15.6
3.	Spleenomegaly	4	7.8
4.	Ascites	2	3.9

8) ANALYSIS OF ASSOCIATED COMPLICATIONS

Anaemia was the most common complication followed by pre eclampsia.

TABLE – 8

Sl.No.	Complication	No. of Cases	Percentage
1.	Anaemia	14	27.4
2.	Pre-eclampsia	4	7.8
3.	Encephalopathy	2	3.9
4.	Hepatorenal failure	1	1.9
5.	Post Partum Haemorrhage	10	19.6

6.	Coagulation failure	2	3.9

9) SERUM BILLIRUBIN LEVEL

In more than 70% of patients, the level of serum billirubin is in the range of 2-6mg%. None of the patients showed serum billirubin level above 20mg%.

TABLE - 9

Sl.No.	Serum Billirubin (mg/dl)	No. of cases	Percentage
1.	2-4	15	29.4
2.	4-6	23	45
3.	6-10	8	15.6
4.	10-14	2	3.9
5.	> 14	3	5.8
	Total	51	100

10) SERUM TRANSAMINASE LEVEL

More than 50% of patients had serum transminase level between 100-400 IU/L.

TABLE – 10

Sl.No.	Serum Transaminase (IU/L)	No. of Cases	Percentage
1.	< 100	19	37.2
2.	100-400	29	56.8

3.	> 400	3	5.8
	Total	51	100

11) ANALYSIS OF FETAL OUTCOME OUT OF 51 CASES

Fetal outcome was determined based on abortion, term live birth, term neonatal death, term IUD and preterm deliveries.

Total number of fetal death is 13.

Total number of babies discharged alive is 35 including preterm and term babies.

Perinatal mortality is 25.4%.

TABLE - 11

Sl.No.	Fetal Outcome	No. of Cases	Percentage
1.	**Abortion**	**2**	**3.9**
2.	**Term Deliveries**	**28**	**54.9**
	Term Alive	24	50.9
	Term IUD	1	1.9
	Term neonatal death	1	1.9
3.	**Preterm deliveries**	**21**	**41.1**
	IUD	3	5.8
	Stillborn	1	1.9

	Neonatal death	7	13.7
	Discharged alive	11	21.5

12) ANALYSIS OF BIRTH WEIGHT AND FETAL OUTCOME

Around 60% of babies were in birth weight between 2-3 kg. Most of the fetal death (90%) were in birth weight group of 1-2 kg. The influence of birth weight on fetal outcome is shown in Table -12.

TABLE – 12.

Sl.No.	Baby weight in Kg	No. of Cases	Percentage	Alive	Death
1.	1-1.5	6	11.76	0	6
2.	1.6-2	9	17.64	4	5
3.	2.1-2.5	15	29.4	15	-
4.	2.6-3	15	29.4	14	1
5.	3.1-3.5	2	3.9	1	1
6.	> 3.5	2	3.9	2	-

13) MATERNAL OUTCOME OF 51 CASES (From January 2004 to August 2006).

TABLE - 13

1.	Total number antenatal admissions	40219
2.	Total number of maternal mortality	25
3.	Incidence of maternal mortality	62/100,000

4.	Total number of jaundiced cases	51
5.	Total number of mortality due to jaundice	6
6.	Percentage of mortality in jaundice	11.76
7.	Percentage of mortality due to jaundice in relation to total maternal mortality	23%

14) RELATION OF MATERNAL AGE TO MATERNAL MORTALITY

Maternal mortality was found to be higher in those women between 20-24 years (50%). Age group wise analysis of maternal mortality is given in Table 14.

TABLE – 14

S.No.	Age group in years	No. of cases	Maternal Mortality	% of maternal mortality in age group.
1.	15-19	2	1	16.6
2.	20-24	30	3	50
3.	25-29	15	2	33.3
4.	30-35	4	-	-
	Total	51	6	100

15) MATERNAL MORTALITY IN RELATION TO GRAVIDITY

Gravidity wise analysis revealed more number of mortality occurred in primigravida (66%). The results are shown in Table 15.

TABLE - 15

Sl. No.	Gravidity	No. of cases	Maternal mortality	Percentage
1.	Primigravida	30	4	66.6
2.	Second gravida	12	1	16.6
3.	Third gravida	8	1	16.6
4.	Fourth gravida	1	-	-
	Total	51	6	100

16) RELATION OF SERUM BILLIRUBIN LEVEL TO MATERNAL MORTALITY

50% of maternal mortality occurred with serum billirubin above 10mg/dl. However 16% of maternal mortality occurred with Billirubin levels of 2-4mg/dl.

TABLE – 16

Sl.No.	Serum Billirubin mg/dl	No. of cases	Maternal Mortality	Percentage of maternal mortality
1.	2-4	15	1	16.6
2.	4-6	23	1	16.6
3.	6-10	8	1	16.6
4.	10-14	2	1	16.6
5.	>14	3	2	33.3
	Total	51	6	100

17) RELATION OF SERUM TRANSAMINASE LEVELS TO MATERNAL MORTALITY

Most of the maternal mortality was observed with value of above 100 IU/L.

TABLE – 17

Sl.No.	Serum Transaminase IU / L	No. of cases	Maternal Mortality	Percentage
1.	< 100	19	1	16.6
2.	100-400	29	3	50
3.	>400	3	2	33.3
	Total	51	6	100

18) RELATION OF TYPE OF DISEASE TO MATERNAL MORTALITY

66.6% of maternal mortality were due to viral hepatitis. Among these 50% died of Hepatitis B infection.

TABLE – 18

Sl. No.	**Diagnosis**	**No. of Cases**	**Maternal Mortality**	**Percentage of Maternal mortality**
1.	Viral Hepatitis	35	4	66.6
	Hepatitis B	11	1	
	Hepatitis B+C	2	1	

2.	HELLP Syndrome	4	2	33.3
3.	Chronic liver disease and PHT	3	0	0
4.	Intrahepatic cholestasis of pregnancy	2	0	0
5.	Malaria	2	0	0
6.	Leptospirosis	2	0	0
7.	Typhoid	1	0	0
8.	Combined malaria and leptospirosis	1	0	0
9.	Gilbert's disease	1	0	0

19) RELATION OF MODE OF DELIVERY TO MATERNAL MORTALITY

Mode of delivery did not alter the maternal outcome according to this study.

TABLE – 19

Sl.No.	Mode of delivery	No. of cases	Percentage	Maternal mortality	% of maternal mortality
1.	Labour natural	43	84.3	3	50
2.	LSCS	5	9.8	2	33.3
3.	Caesarian	1	1.9	1	16.6

	hysterectomy				
4.	Abortion	2	3.9	0	0

20) CAUSES OF MATERNAL MORTALITY

The detailed analysis of various causes of maternal death is shown in Table 20.

TABLE – 20.

Sl. No.	Causes of Maternal Mortality	No. of cases	Percentage of maternal Mortality
1.	Post partum haemorrhage	1	16.6
2.	Hepatorenal failure	1	16.6
3.	Hepatic coma	2	33.3
4.	Coagulation failure	2	33.3

DISCUSSION

The incidence of jaundice among the pregnant women attending the Govt. R.S.R.M. Lying-in Hospital, Royapuram, Chennai from January 2004 to August 2007 is 1.26 per 1000 popultion. The incidence in various places by various authors is shown in the following table.

Sl. No.	Author	Year	Incidence per 1000 population.
1.	Bhaskar Rao (Madras)	1955	0.26
2.	Sarkar et al (Calcutta)	1992	2.3
3.	Reddi Rani et al (Pondicherry)	1993	1.17
4.	Devinder Kaur et al (Delhi)	2001	0.92

Our hospital incidence correlates with the study of **Reddi Rani et al**, 1993. Our hospital incidence is very high when compared to Western countries where it is 1 in 1500 pregnancies **(Williams 1996)**. This is because of higher prevalence of malnutrition, poor sanitation and low socioeconomic status in our country.

ETIOLOGY

In the present study viral hepatitis was found to be the commonest cause (68.5%) next being HELLP syndrome (7.8%) third in order is chronic liver disease (5.8%). Other causes are intrahepatic cholestasis of pregnancy

(3.9%), malaria (3.9%), leptospirosis (3.9%), typhoid (1.9%) and Gilbert's disease (1.9%).

Patients with viral hepatitis was the major group with Jaundice in pregnancy consisting of 68%. 11 patients had acute hepatitis B infection (21.5%), [2 had combined hepatitis B & C infection (4%)] and 24 were presumptively taken as type E (47%). Jaiswal SP and Naik G in 2001 and Jain A, Sharma JK in 1999 analysed various types of viral hepatitis during pregnancy. Their studies were shown below :

Place	**Author**	**Year**	**HAV**	**HBV**	**HCV**	**HDV**	**HEV**	**No viral markers**
Indore	Jaiswal SP Naik G Soni N	2001	7%	19%	4%	Nil	46%	24%
New Delhi	Jain A Sharma JK	1999	4%	28%	2%	Nil	42%	18%

All HCV infected patients were coinfected with other hepatotropic viruses and the most common coinfecting agent was found to be HBV. The prevalence of HAV, HCV and HDV were very low in the above studies.

Because of universal exposure during infancy and childhood, HAV infection is more common in children rather than in adults in developing

countries like India. HDV always coinfects with Hepatitis B virus and cannot survive independently. Hence acute viral hepatitis in an adult in a high prevalence country such as India is more likely to be HEV or HBV rather than HAV, HCV or HDV **(Michael de Swiet, 2002).**

AGE :

In the present study 58.8% of pregnant jaundiced women were between 20-24 years. This correlates with the study of **Sheth Abhay et al, 1990, Devinder Kaur et al, 2001 and Kamala Jeyaram et al, 1988**. This age group is having the maximum fertility rate and maximum number of deliveries. Early age of marriage in lower socioeconomic group due to illiteracy also contributes. (In Western Countries, only 30% are under 24 years).

GRAVIDITY

In this study 59% of patients were primigravida; It correlates with the study of **Reddi Rani et al, 1993** and **J.S. Chauhan et al, 1983** (50%). **Kamala Jeyaram et al, 1986** and **Devinder Kaur et al, 2001** had showed only 30% incidence among primigravida.

GESTATIONAL AGE

76.4% of patients presented in 3rd trimester. This correlates well with the study of **C.M. Alwani et al., 1986**, **Reddi Rani et al, 1993**. Increaed incidence in third trimester is probably due to excess nutritional stress in late pregnancy.

SOCIOECONOMIC STATUS

95% patients belonged to lower socioeconomic group in this study. This shows the influence of malnutrition, poor sanitation, water contamination, sexual abuse and poor health awareness in the development of jaundice as stressed by various authors.

SIGNS AND SYMPTOMS

Analysis of symptoms shows majority of patients had fever (71%) followed by Anorexia, nausea and vomiting (55%). This correlates with the study of **Subodh Singh R, Chauhan et al, 1991** and **Jai Bagwan sharma et al, 1990**. In our study 96% of patients presented with jaundice. This is comparable with study of **Kamala Jeyaram et al, 1988** (93%). But **Sunanda Kulkarni, 1996** has reported jaundice in only 60% of cases. Hepatomegaly was present in 16% of cases.

BIOCHEMICAL PARAMETERS

In this study, 57% of maternal mortality were with serum billirubin more than 10mg/dl. In the study of **Chadda et al, 1983** majority of mortality occurred with serum billirubin level more than 15mg. 50% of maternal mortality were observed with serum transaminase levels between 100-400IU/L in our study. Mortality also reported with low level and this is due to exhaustion by previous excessive release of transaminase **(Issel Backer 1987)**.

The prothrombin time is the most sensitive indicator of severity of liver dysfunction and hence the prognosis (**Weinstein et al, 1982**). A mildly elevated prothombin time usually indicates concurrent DIC whereas grossly elevated prothombin time signify significant hepatic necrosis. **Khuroo M.S. & Kamili** in 2003 reported poor prognosis in patients with prothombin time > 30 secs. In our study out of 6 maternal deaths 4 patients had prothombin time > 30 secs, one had 26 sec and other had 22 sec.

COMPLICATION

14 patients had anaemia. Among them 2 had severe anaemia (Hb < 7gm%) and rest of them were between 7-10gm%. Among the 2, one patient

died due to hepatic coma and other survived, but baby died within 24 hours of delivery. Among 4 pre-eclampsia patients 2 died. Anaemia and pre-eclampsia in a jaundiced patient further worsens the prognosis.

10 patients had PPH. 2 patients died of PPH (One had atonic PPH and other was due to DIC). Among 10 patients, 8 required blood transfusion. **Alwani et al, 1985** reported 31% incidence of post portum haemorrhage in his studies. Our study has significantly lower incidence of PPH (20%).

MODE OF DELIVERY

10% of patients were delivered by LSCS for obstetric indications and 85% had labour natural. Since most were preterm, labour was easier. Mode of delivery did not influence maternal or fetal outcome.

Early delivery by caesarian section to improve maternal and fetal survival and arresting the disease is recommended by **Peter's et al, 1967** and **Burrough's et al., 1983**. However the risk of anaesthesia and bleeding due to coagulation abnormalities are the factors against it.

FETAL OUTCOME

Among the total 51 patients, 4% had abortion, 41% had preterm delivery and 55% had term deliveries. **Sheth Abhay et al, 1999** reported 3% incidences of abortion and 55% preterm deliveries whereas **Subodh Singh et al, 1991** reported 8% abortion and 64% preterm delivery.

Total number of fetal death is 13. Among these 11 were preterm babies and only 2 was term. All neonatal deaths were because of prematurity and low brith weight. Prematurity and low brith weight as a cause of neonatal mortality was stressed by various authors (**Jai Bagwan Singh, 1990, Subodh Singh et al, 1991, Devinder Kaur et al, 2001**). Fetal loss by abortion and prematurity may be due to high fever and general debility associated with high viraemia. Perinatal mortality is 24%.

MATERNAL OUTOME

Out of 51 jaundiced pregnant women in this study, 6 women died. 2 patients died of HELLP syndrome, 4 patients due to viral hepatitis. Among the 4 patients, one was positive for HBsAg and another was positive for both HBsAg and anti HCV.

HELLP Syndrome

HELLP Syndrome patients with jaundice constituted 4 patients, all of whom presented in late second and early 3rd trimester with severe hypertension. **Weinstein et al, 1985** has reported the mean gestational age of presentation of HELLP syndrome was 33.6 wks.

Among 4 patients, 3 were preterm delivery and one was intrauterine death. Perinatal mortality in this group was 50% (2 out of 4). **Weinstein et al, 1985, Sibai et al, 1990** has reported a perinatal mortality rate of 60% in patients with HELLP syndrome. Higher perinatal mortality is due to prematurity. There was 2 maternal deaths in this group. One patient died due to acute renal failure. Though her pregnancy was terminated by preterm LSCS, patient didn't recover. Another patient, whose pregnancy was terminated for IUD developed DIC. In spite of massive blood transfusion patient died. **Baha et al, 1990**, reported a maternal mortality of 24% in his study, but it was high in our study (50%).

Viral Hepatitis :

Out of 35 patients due to viral hepatitis, there were 4 maternal deaths. One patient had acute hepatitis B infection and she presented with

term IUD. Her pregnancy was terminated by LSCS due to failed induction but proceeded to caesarian hysterectomy due to atonic PPH. Her prothrombin time was prolonged. Another patient had combined hepatitis B & C infection and she died of fulminant hepatic failure. The other 2 patients were negative for these 2 viral markers. One patient died of hepatic coma and she had preterm delivery. Another had LSCS done but died on I POD due to DIC. Both babies were alive.

Maternal mortality in this group was 12%. Maternal mortality among pregnant women with viral hepatitis was 14.3% whereas is nonpregnant women it was 5.6% **(De Swiet 2002)**. Age and parity did not reveal any significance.

In the studies reported by **Alwani et al, 1985** the cause of death among patients with viral hepatitis was hepatic coma in 2.4%, GI bleed in 15%, Hepatorenal syndrome in 15%, PPH in 13% and Hepatic encephalopathy in 8%. **Mirghani et al, 1992**, observed more than 80% of deaths occurred in postpartum period. It is concluded that pregnancy is a risk factor which increases the mortality of viral hepatitis.

In the present study 63% had term delivery, 31.4% had preterm delivery and 5.7% had IUD. Perinatal mortality calculated in this group was 25.7%. Observation in other studies includes,

Author	Year	Abortion	Preterm delivery	Perinatal Mortality
Medha et al	1993	8.3%	20%	27%
Padmaker	1986	8.3%	24%	30.9%
Alwani et al	1985	11.9%	18%	29%

IHCP :

There were 2 patients with intrahepatic cholestasis of pregnancy in the study. Both presented with pruritis and Jaundice for 2-3 wks duration. There was no clinical evidence of viral hepatitis in these patients. One patient had term delivery and another one was borderline term. Both babies were alive (**Rioseco AJ, 1994)** has reported an increased incidence of preterm labor (44% and 50% respectively) in IHCP patients. But maternal and perinatal outcome was good in our study.

Cirrhosis and Portal Hypertension :

In 3 patients with chronic liver diseases, one had chronic active hepatitis due to combined Hepatitis B & C infection. She had oesophageal

devascularisation done for variceal bleed 2 years before conception. She was on T.lamivudine 100mg bd throughout her pregnancy. She had uneventful term delivery and baby was alive.

Another patient had cirrhosis and ascites and she landed in abortion. One patient with non cirrhotic PHT had preterm delivery but baby was alive. There was no maternal and perinatal mortality in this group except for one abortion.

Maternal mortality is 4-7% in NCPHT whereas it is 10-18% in cirrhosis PHT **(Shriram PV, Goenka MK, 1999)**. Perinatal mortality in cirrhotic patient is 10 to 28% **(Schreyer, P, 1982)**.

Other Causes :

One patient had combined malaria and leptospirosis. She presented at 22 weeks of pregnancy with H/o fever and vomiting for 1 week and Jaundice for 3 days. Her serum Billirubin was 4 mg%. She was intensively treated with T. chloroquine and inj. Crystalline penicillin. She recovered in 2 weeks and was discharged. At term she was readmitted in our hospital and had full term normal delivery without any complications.

One patient with jaundice was widal positive and she presented at 26 wks with H/o. fever, anorexia and loose stools for 10 days. Her serum billirubin was 3.8mg% and transaminase was elevated. She had preterm delivery but baby died due to low birth weight. Mother recovered.

Two patients with jaundice and leptospira positive presented at II trimester. Serum billirubin was more than 10mg% in both patients. One patient had abortion and other had stillbirth. Both mother were intensively treated and recovered. Perinatal mortality is high in leptospirosis. 30% of pregnancies ended in abortion or preterm death **(Shaked Y, Samra, D, 1993)**.

Two patients with fever, jaundice and spleenomegaly were positive for malarial parasites. One patient had preterm labour and baby survived whereas the other patient had IUD during the treatment but mother recovered well. The incidence of preterm delivery, abortion, stillbirth and intrauterine deaths are frequent in malaria in pregnancy **(15-60% by Brabin et al, 1990).** Maternal outcome is not so much affected except in cerebral malaria where maternal mortality is 40%.

One patient who was diagnosed as Gilbert's disease in previous pregnancy presented to us at 38 wks with H/o. jaundice and pruritis on a off

for past 5 months. Her billirubin level was 4mg%. She had uneventful delivery and baby was alive.

MANAGEMENT

In patients with HELLP syndrome immediate termination of pregnancy once diagnosed is the treatment of choice. Strict control and frequent monitoring of BP is mandatory. In IHCP, oral antihistaminic may provide some relief from pruritis, but UVCA is the preferred treatment. If patient is anicteric, pregnancy can be allowed to continue till term, otherwise pregnancy has to be terminated after completion of 36 weeks.

Management of patients with viral hepatitis was entirely symptomatic and supportive. It includes bed rest, high carbohydrate diet, glucose, vitamin B complex and IV dextrose. In none cases, termination of pregnancy was carried out prophylactically. Operative interference was restricted to minimum. Tab. Chloroquine was given to patients with malarial hepatopathy. Patients with leptospirosis was treated with crystalline penicillin. Patients with typhoid fever was given Tab. Ciprofloxacin. Patients with chronic liver disease did not require any specific therapy except for frequent monitoring of liver function tests and

coagulation profile. Blood and fresh frozen plasma were given when appropriate.

FETAL ANOMALIES

There was no congenital anomaly in the present study. **Shah** had reported that there is no convincing evidence that jaundice in the I trimester can cause fetal anomalies. So jaundice is not an indication for medical termination of pregnancies.

DEVELOPMENT OF NEONTAL JAUNDICE

4 babies had only physiological jaundice. A jaundiced mother giving birth to a jaundiced baby is rare because in most cases, conjucated billirubin is present to which placenta is impermeable (**Sechar**).

TRANSPLACENTAL TRANSMISSION OF HBsAG

The cord blood of 11 babies of HBsAg positive mothers were tested for HBsAg. All but one were negative for HBsAg. All babies received immunoglobulin and vaccination.

SUMMARY

1) Incidence of jaundice in R.S.R.M. Lying-in Hosptial, Chennai during the year 2004 to 2006 was 1.26 per 1000 pregnant women. Total number of cases were 51.

2) Viral hepatitis is the commonest cause (35 cases constituting 68%)

3) Out of these 35 cases of viral hepatitis, 11 had hepatitis B infection (2 had combined hepatitis B & C infection).

4) 58.8% were in 20-24 year age group. Fulminant hepatic failure is also higher in this age group.

5) Primigravidas constituted 58%.

6) The maximum incidence was in III trimester (76.4%).

7) The mortality was more in puerperium.

8) These people belonged to lower socioeconomic group.

9) Maternal and fetal prognosis were worse with high levels of serum billirubin, but prognosis was affected by low levels also.

10) Very high levels of serum transaminase indicate marked hepatocelluular damage and bad prognosis. Sudden fall to low levels is also ominous.

11) Serum alkaline phosphatase levels were raised to high levels in intrahepatic and extrahepatic cholestasis. It did not correlate with the prognosis.

12) Serum prothombin time is a good indicator of prognosis. PT of > 30 secs indicates worse prognosis.

13) Anaemia and PIH in a pregnant jaundiced women worsens the prognosis.

14) Maternal and perinatal outcome was good in intrahepatic cholestasis of pregnancy.

15) In HELLP syndrome with jaundice maternal and perinatal mortality was 50% respectively.

16) Perinatal mortality is high in jaundice due to malaria, leptospirosis and typhoid.

17) No significance could be given to the mode of delivery in relation to maternal and fetal outcome.

18) Incidence of preterm delivery is 41.1%

19) 60% of babies were weighting < 2.5kg.

20) Perinatal mortality in pregnancies complicated by Jaundice is 25.4% (254/1000 livebirth).

21) Low birth weight and prematurity predisposed to high fetal loss.

22) Maternal mortality among jaundiced patients is 11.76%.

23) Viral hepatitis contributes maximum to the maternal mortality (66.6%). Two deaths were due to Hepatitis B infection.

CONCLUSION

Jaundice in pregnancy may be caused by several factors. But in developing countries like India, the commonest cause is viral hepatitis (as proved in the study), mostly due to waterborne non B type (HEV). Next in frequency is followed by HBV and then HCV. Acute viral hepatitis in an adult in a high prevalence country such as India is most likely to be due to HEV or HBV rather than HAV or other viruses.

The maternal mortality in pregnant women with viral hepatitis is 14.3% and in non-pregnant women it is 5.6% indicating a high relative risk of death (4:1) for pregnancy. The high maternal and fetal wastage attributable to viral hepatitis clearly shows that the problem of viral hepatitis in pregnancy needs to be addressed.

Anaemia, pre-eclampsia and post partum haemorrhage in a jaundiced pregnant women carries worst prognosis. Because of high endemicity, malaria and leptospirosis also contributed significantly to the cause for jaundice in our study. Perinatal mortality were high in this group.

Perinatal mortality in jaundice complicating pregnancy is mainly due to prematurity and low birth weight. Other major area of concern is the risk

of vertical transmission of HBV. This can be greatly minimized by giving HBV immunoglobulin and vaccine to the newborn immediately after delivery. Vertical transmission of HCV is rare unless there is HIV coinfection or high degree of viraemia.

To conclude all pregnant women should be screened for HBsAG as a routine. Health education, good hygiene, provision of safe water and improvement of basic sanitation will help to prevent disease transmission. To reduce the incidence of hepatitis B at the root level, Hepatitis B vaccination should be given to all newborn.

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PROFORMA

S.No.:

Name: Age:

Unit: I.P.No. DOA: DOD:

Address:

LMP: EDD:

G: P: L: A:

COMPLIANTS ***Duration***

H/O Pain :

H/O Bleeding PV :

H/O Draining PV :

H/O Nausea, Vomiting :

Anorexia :

Purities :

Malaise

H/O Fever

GI Bleed

Jaundice

Oliguria

Haemetemesis

Edema legs

Altered Sensorium/Loss of Consciousness

Indigenous Treatement : Tab./Inj.

GENERAL EXAMINATION

Built		Pulse:	BP:
Anemia		CVS:	RS:
Icterus	CNS:	Higher Funtion	
Fever		Hltered Sensorium	
PE		Flap	
Spider Naevi	Abdomen		
Palmar erythema		Ascitis	
Flap		Distended Veins	
		Hepatomegalay	

Splenomegaly

OBS. EXAMNIATION

PA: Uterus: PV:

Presenting Part:

FH:

INVESTIGATION

Urine: Albumin/Sugar/Deposits C/S:

BS/BP: Urobilinogen:

Blood Grouping: Complete

Hemogram:

BT: CT: Peripheral Smear: Platelet Count:

Blood Urea: Blood Sugar: Sr. creatinine:

Sr. Uric Acid:

LFT: SGOT/SGPT/SAP

Total Proteins/Albumin/ Globulin:

Sr. Viral Markers :

Sr. Fibrinogen :

VDRL :

Sr. Electrolytes :

Blood for leptospirosis :

OBS.OUTCOME

Abortion/IUD/Still Birth/Preterm/Term:

Nature of delivery

Labour Natural/Labour Natural with episiotomy

Forceps/Vacuum/LSCS

Third Stage of Labour

PPH:

BABY DETAILS:

Baby Sex/Wt/Apgar/Jaundice/Cord Blood.

TREATMENTS GIVEN

IV Fluids/Antibiotics/Diuretics/Inj.Vitamin K/Others

Blood Transfusion/FFP

CLINICAL DIAGNOSIS

MATERNAL OUTCOME

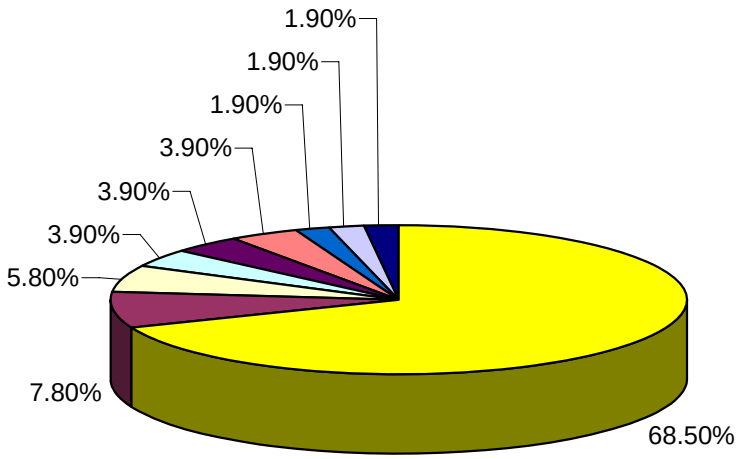
FETAL OUTCOME

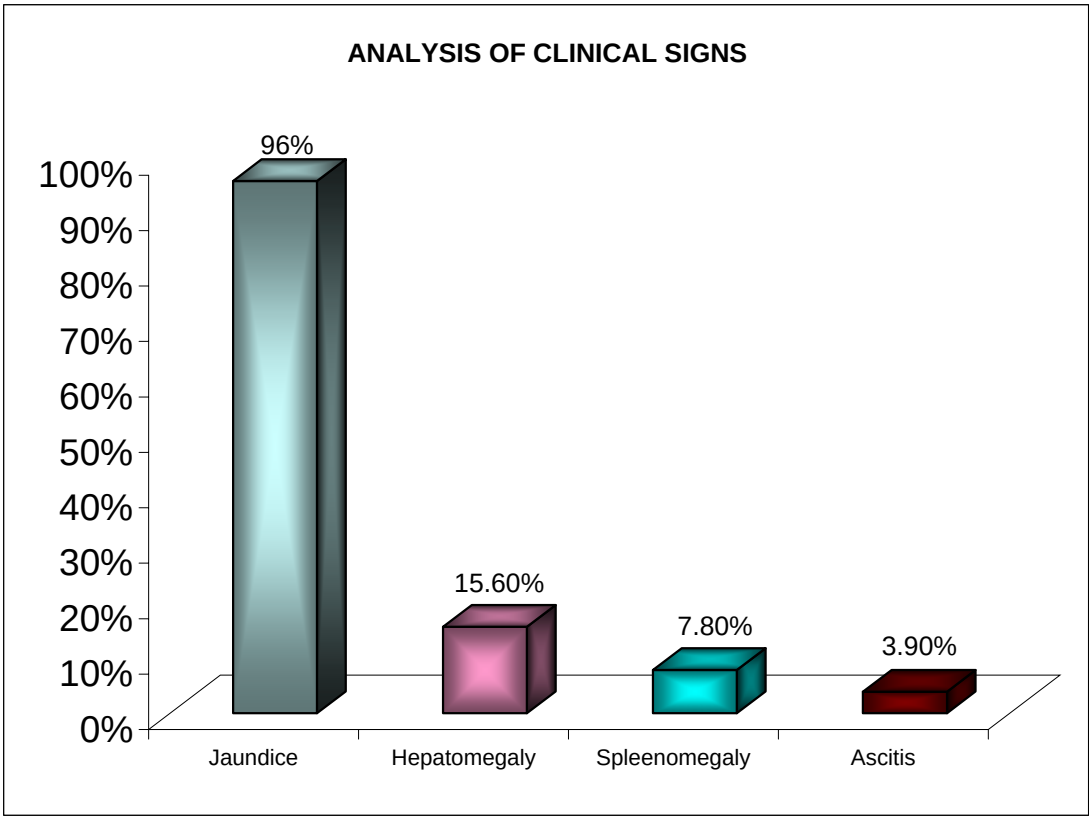
MASTER CHART

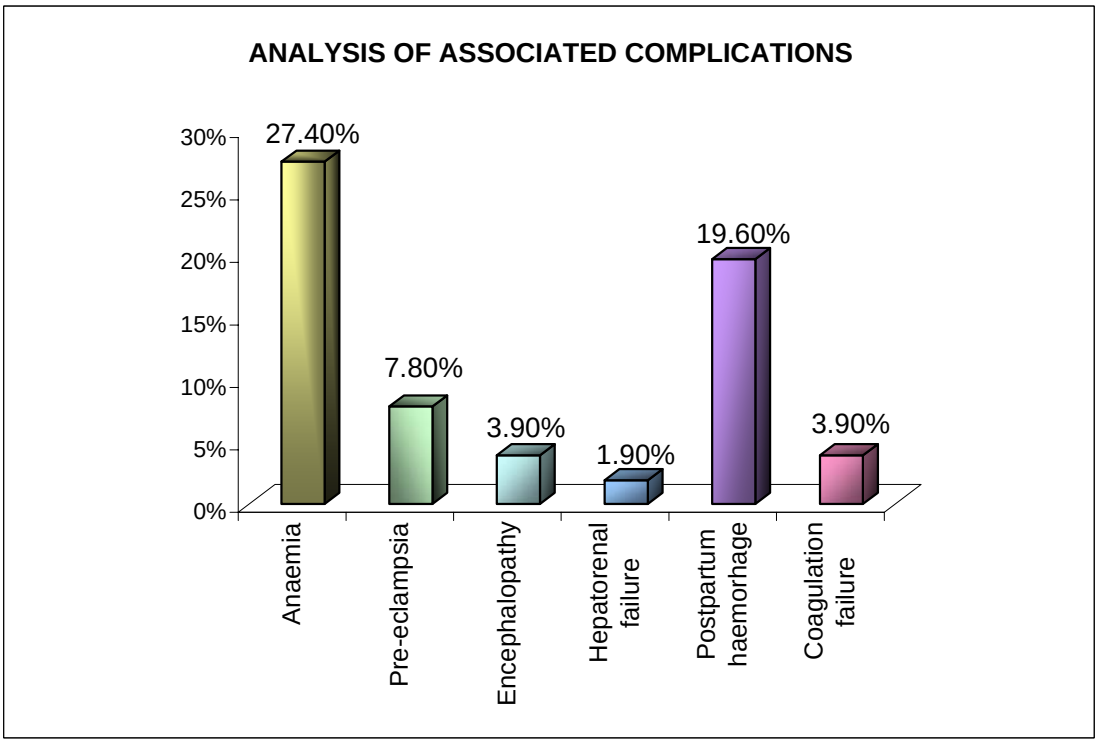
S.No.	Name	Age	O/H	GA in Wks.	Symptoms	Duration	Signs	Complications	U/A	Urine BS/BP	BT	CT	PT	Hb	Platelets	Sr.Billirubin	SGPT	SAP	HBsAg	Anti-HCV	P.Smear	Widal	Leptospirosis	FO	MOD	Bady Wt.	Mother	PPH	Cause
1	Vinotha	27	G2P1L1	34	1,2	1Week	1	1,2,3	++	-	8min	14min	26sec	7.4	85000	4.6	260	217	-	-	-	-	-	PT	LSCS	2.1	D	-	HRF
2	Latha	20	Primi	40	2,3,5	3days	1	-	-	+	N	N	N	11	2.4	5	110	200	-	-	-	-	-	Term	LN	2.5	A	-	-
3	Sasikala	24	G2P1L1	36	1,2,3,6	17days	1,3	-	+	+	N	N	N	9	2.1	6	80	140	-	-	MP+	-	-	PT	LN	1.9	A	-	-
4	Indumathi	20	G3P1L1A1	29	3,5	10days	1,2,3	1	-	+	N	N	N	8	1.75	8	140	186	-	-	MP+	-	-	IUD	LN	1.7	A	-	-
5	Sathiya	22	Primi	37	1,2,3,7	4days	-	1	-	+	N	N	N	8.5	2	4	110	140	+	-	-	-	-	Term	LN	2.8	A	+	-
6	Kala	23	Primi	36	5,6,9	25days	1	-	-	+	N	N	N	10	2.1	6	135	652	-	-	-	-	-	B. Term	LN	2.2	A	-	-
7	Govindammal	25	G3P2L2	26	1,2,3,7	10days	1	-	-	+	N	N	N	9	2.4	2.8	80	317	-	-	-	+	-	PT-ND	LN	1.3	A	-	-
8	Lathifa	26	Primi	37	3,4	1Week	1	-	-	+	N	N	N	12	2.2	6	128	113	+	-	-	-	-	Term	LN	3	A	-	-
9	Revathi	29	Primi	36	3,8	12days	1	-	-	+	N	N	N	11	2.1	2.5	60	90	-	-	-	-	-	Term	LN	2.5	A	-	-
10	Kavitha	22	G2P1L1	26	5,8	8days	1,2,4	-	-	+	N	11min	21sec	10	2.1	4.5	120	170	-	-	-	-	-	PT-ND	LN	1.4	A	-	-
11	Santhi	32	G3P2L2	40	2,3	3days	1	1	-	+	N	N	N	6.8	2.1	7	160	146	+	-	-	-	-	Term-ND	LN	2.8	A	+	-
12	Manjula	24	Primi	34	1,3,5	7days	1,2	1	-	+	N	10min	19sec	8	2.4	6	80	96	+	-	-	-	-	PT-ND	LN	1.8	A	+	-
13	Veeralakshmi	19	Primi	34	4	1Week	1,2,5	1	-	+	N	13min	46sec	6	2.1	2.8	518	255	-	-	-	-	-	PT	LN	1.8	D	-	Hepatic coma
14	Leema	25	G2P1L1	27	1,2,3,4,(	6days	1	-	+	+	N	N	N	9.2	2	16	250	117	-	-	-	-	+	Stillborn	LN	1.3	A	-	-
15	Desammal	20	G2P1L1	20	1,3	5days	1	-	-	+	N	N	N	9.2	1.8	4	60	113	-	-	MP+	-	+	Term	LN	2.7	A	-	-
16	Tamilselvi	21	Primi	38	1,2	1 Month	1	4	-	+	N	N	17Sec	10	2.2	4	52	125	+	+	-	-	-	Term	LN	2.7	A	-	-
17	Prema	32	G3P2L2	22	2,4,6	20days	1,3,4	1,5	-	-	N	N	N	7	1.7	6	92	140	-	-	-	-	-	Abortion	-	-	A	-	-
18	Ammu	29	Primi	38	5,6,9	1 Month	1	-	-	+	N	N	N	11	2.1	4.5	110	550	-	-	-	-	-	Term	LN	3	A	-	-
19	Usha	29	Primi	36	2	1days	1	2	+++	+	2min	7min	N	11	95000	2.8	96	112	-	-	-	-	-	PT	LN	2	A	+	-
20	Jayanthi	24	Primi	40	2,4	2days	1	-	-	-	6min	20min	32sec	10	1.3	6.2	132	217	-	-	-	-	-	Term	LSCS	2.8	D	-	DIC
21	Rathidevi	23	Primi	39	1,3,5	4days	1	-	-	+	N	N	N	11	2.3	9	120	196	+	-	-	-	-	Term	LN	2.3	A	-	-
22	Vasantha	22	G2P1L1	28	1,2	3days	1	2	++	+	N	N	N	9	100000	3	484	207	-	-	-	-	-	PT-ND	LN	1.5	A	-	-
23	Rani	23	Primi	34	1,2	3 Months	2,3	5	-	+	N	N	20sec	9.8	2.1	2.6	60	80	-	-	-	-	-	PT	LN	1.8	A	+	-
24	Kalaiselvi	23	Primi	32	1,2,4	2days	1	2	+++	+	6min	>20min	22sec	8.6	65000	18	80	310	-	-	-	-	-	IUD	LN	1.8	D	+	DIC
25	Kaniammal	20	Primi	20	2,3	1Week	1,2	1	-	+	N	N	N	7.8	1.9	10	160	380	-	-	-	-	+	Abortion	-	-	A	-	-

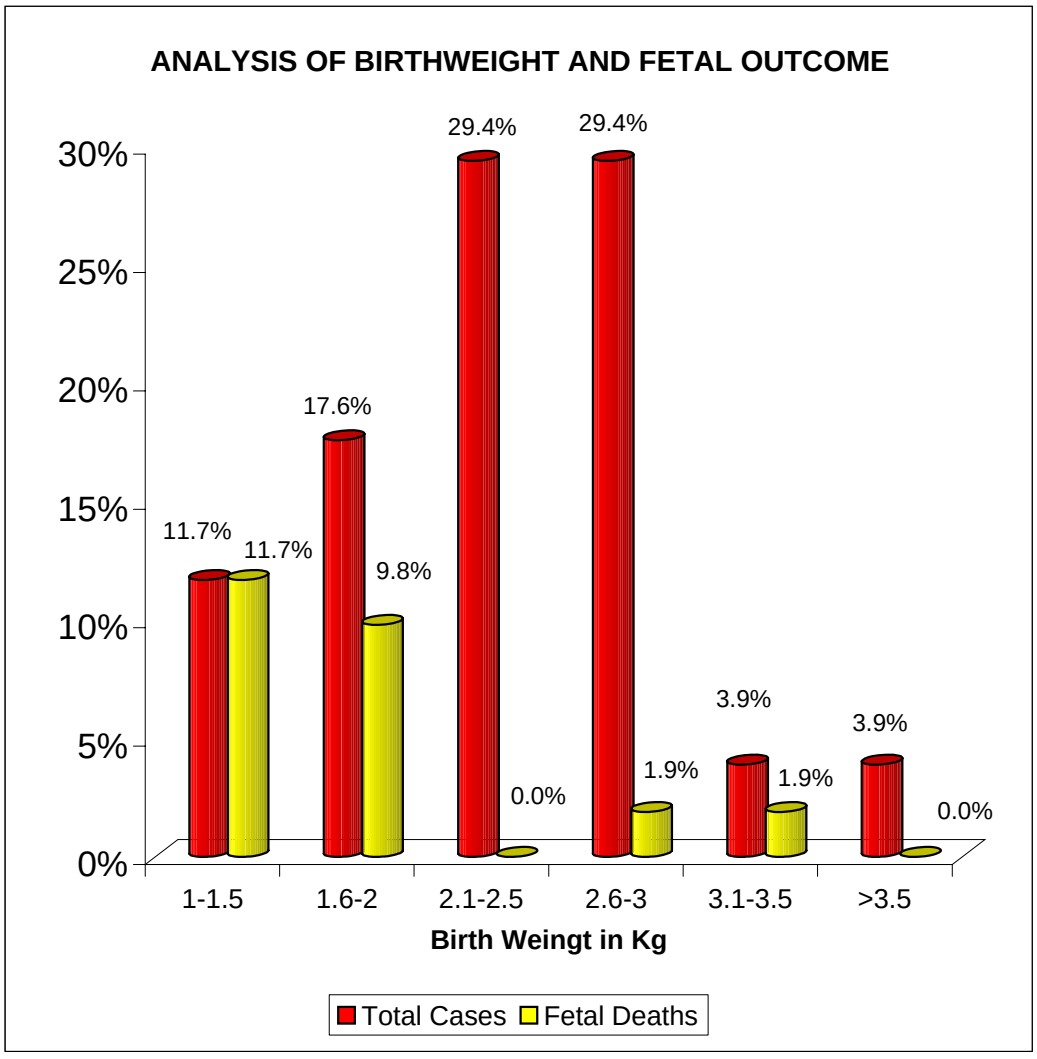
S.No.	Name	Age	O/H	GA in Wks.	Symptoms	Duration	Signs	Complications	U/A	Urine BS/BP	BT	CT	PT	Hb	Platelets	Sr.Billirubin	SGPT	SAP	HBsAg	Anti-HCV	P.Smear	Widal	Leptospirosis	FO	MOD	Bady Wt.	Mother	PPH	Cause
26	Chitra	23	Primi	38	6	5 Months	1	6	-	+	N	N	N	11	2.6	4	30	75	-	-	-	-	-	Term	LN	2.6	A	-	-
27	Naleeni	22	Primi	33	2,3,6	6days	1	-	+	+	N	N	N	12	1.75	12	240	180	+	-	-	-	-	IUD	LN	1.6	A	+	-
28	Vasanthi	34	Primi	38	3,4,5	2days	1	-	-	+	N	N	N	12	2.2	5	126	117	+	-	-	-	-	Term	LSCS	2.9	A	-	-
29	Pushpa	22	Primi	34	1,3,4,5	7days	1	-	-	+	N	N	N	10	2	4	200	115	-	-	-	-	-	PT	LN	2.4	A	-	-
30	Veerama	27	Primi	34	1,3	10days	1	-	-	+	N	N	N	10	2.1	4.8	80	90	-	-	-	-	-	PT	LN	2.5	A	-	-
31	Amsha	27	G2P1L1	38	1,4,6	6days	1	1	-	-	N	N	N	8	2.1	2.5	170	96	-	-	-	-	-	Term	LN	3	A	-	-
32	Selvi	29	G3P2L2	40	1,3,5	9days	1	1	-	+	N	13min	51sec	7	1.85	12.6	430	300	+	-	-	-	-	IUD	LSCS+H	3.1	D	+	Atonic PPH
33	Megala	20	Primi	26	3,5	10days	1	1	-	+	N	N	N	7	2	4	118	260	-	-	-	-	-	PT-ND	LN	1.25	A	+	-
34	Sumathi	26	G2P1L1	35	2,3,5	2days	1	-	-	+	N	N	N	10	1.95	12	178	210	-	-	-	-	-	PT-ND	LN	1.9	A	-	-
35	Uma	27	G2P1L1	39	1,2,3	10days	1	1	-	+	N	N	N	7.8	2.1	5	110	178	+	-	-	-	-	Term	LN	2.2	A	-	-
36	Dilliammal	23	Primi	38	1,3,4,5	3days	1,2	1	-	+	N	15min	59sec	8	1.8	14	226	215	+	+	-	-	-	Term	LN	3	D	-	Acute Liver Failure
37	Padma	35	G3P2L2	40	1,3,4,5	9days	1	-	-	+	N	N	N	10	2.1	4.6	80	90	-	-	-	-	-	Term	LN	3.2	A	-	-
38	Bhavani	22	Primi	38	1,3	5days	1	-	-	+	N	N	N	13	2.5	5	98	110	-	-	-	-	-	Term	LN	2.25	A	-	-
39	Gowri	23	G2P1L1	40	1,3,5	3days	1	-	-	+	N	N	N	10	2	4	80	178	-	-	-	-	-	Term	LN	2.25	A	-	-
40	Jayanthi	22	Primi	36	1,3	4days	1	-	-	+	N	N	N	13	2.2	5	110	136	-	-	-	-	-	Term	LN	2.6	A	-	-
41	Kaniammal	26	G4P3L3	39	1,6	2days	1	-	-	+	N	N	N	9	2.2	5	86	103	-	-	-	-	-	Term	LN	2.4	A	-	-
42	Kanagi	30	G3P2L2	35	1,3,4	7days	1	-	-	+	N	N	N	9.6	2.1	6.1	250	215	-	-	-	-	-	PT	LN	1.9	A	-	-
43	Hema	26	G2P1L1	33	1,3	2days	1	-	+	+	N	N	N	10	2.3	6	86	95	-	-	-	-	-	PT	LN	1.7	A	-	-
44	Kamala	25	Primi	34	1,3,5	6days	1	-	-	+	N	N	18sec	10	2	4	200	210	-	-	-	-	-	PT	LN	2	A	-	-
45	Vadivu	22	G2P1L1	40	1,6	6days	1	-	-	+	N	N	N	9	2.1	3	78	212	-	-	-	-	-	Term	LN	2.9	A	+	-
46	Nirmala	22	Primi	39	3,4,5	3days	1	-	-	+	N	N	N	12	2.4	6	110	176	-	-	-	-	-	Term	LN	2.6	A	-	-
47	Santhi	23	Primi	38	1,3,5	8days	1,2	-	-	+	N	N	N	12	2.2	3.9	140	193	-	-	-	-	-	Term	LN	2.75	A	-	-
48	Jamuna	20	Primi	39	1,3,6	4days	1	-	-	+	N	N	N	10	2.3	4	100	98	-	-	-	-	-	Term	LSCS	3.6	A	-	-
49	Mary	19	Primi	38	1,2,3	4days	1	-	-	+	N	N	N	12	2.6	5.5	105	90	-	-	-	-	-	Term	LSCS	3.7	A	-	-
50	Balkish	21	Primi	41	1,2,3	6days	1	-	-	+	N	N	N	11	2.6	5	118	112	-	-	-	-	-	Term	LN	2.5	A	-	-
51	Meena	24	G2P1L1	27	1,3,6	10days	1	1	-	+	N	N	N	7.4	2.1	5	130	72	-	-	-	-	-	PT-ND	LN	1.1	A	-	-

DISTRIBUTION OF CASES ACCORDING TO AETIOLOGY

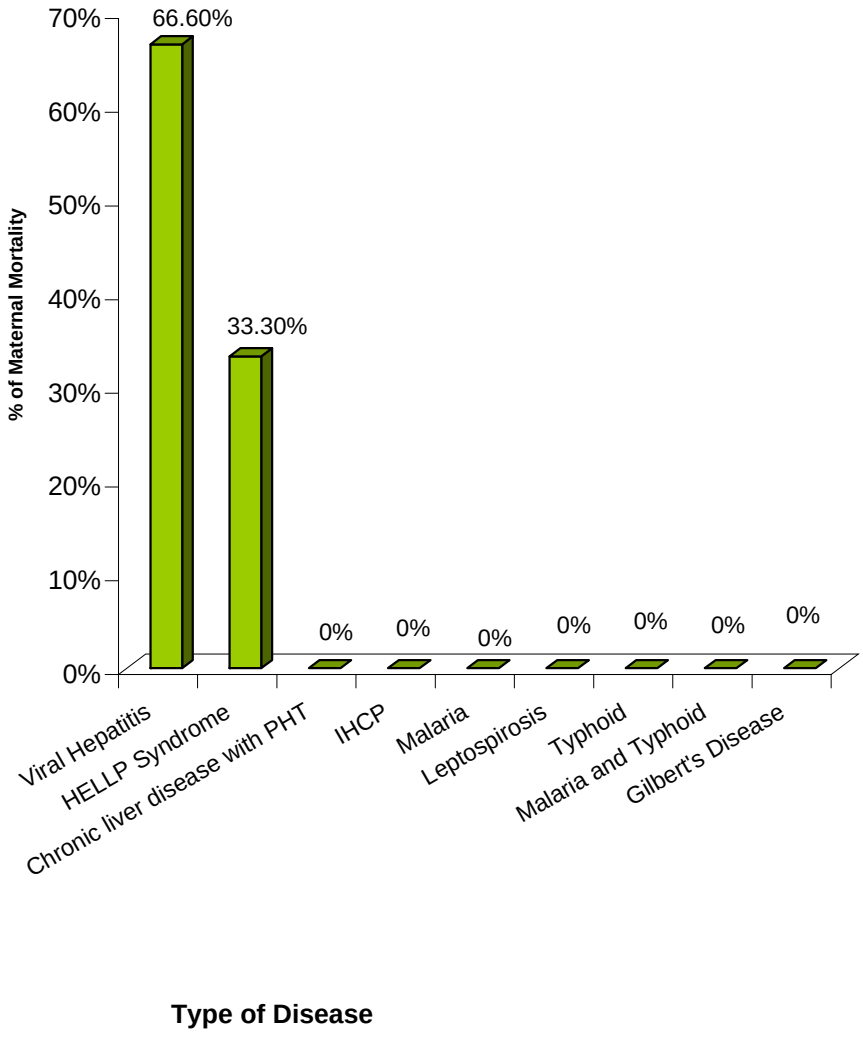


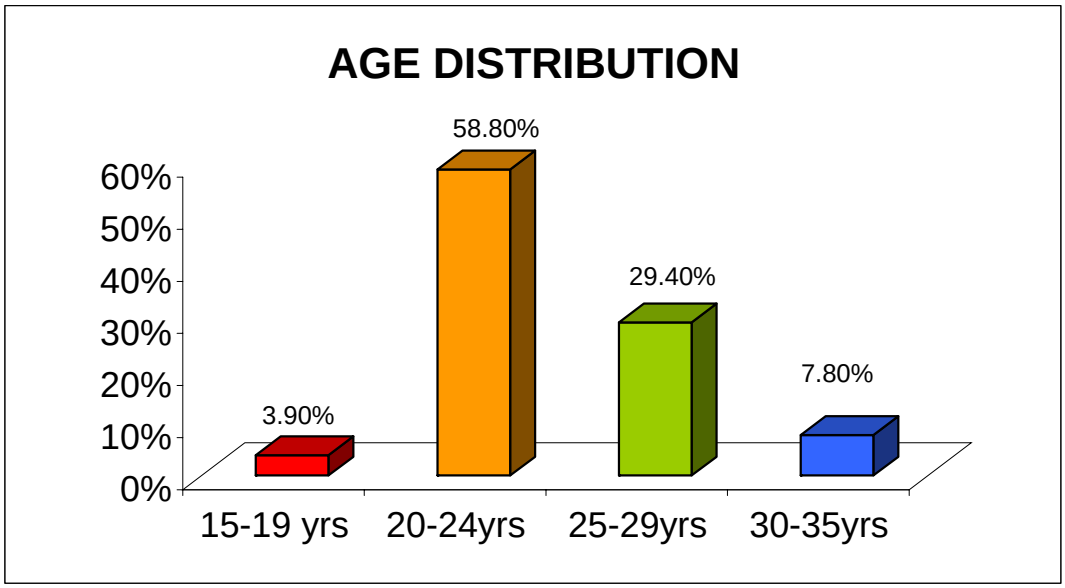

ANALYSIS OF CLINICAL SIGNS
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
96%
15.60%
7.80%
3.90%
Jaundice
Hepatomegaly
Spleenomegaly
Ascitis

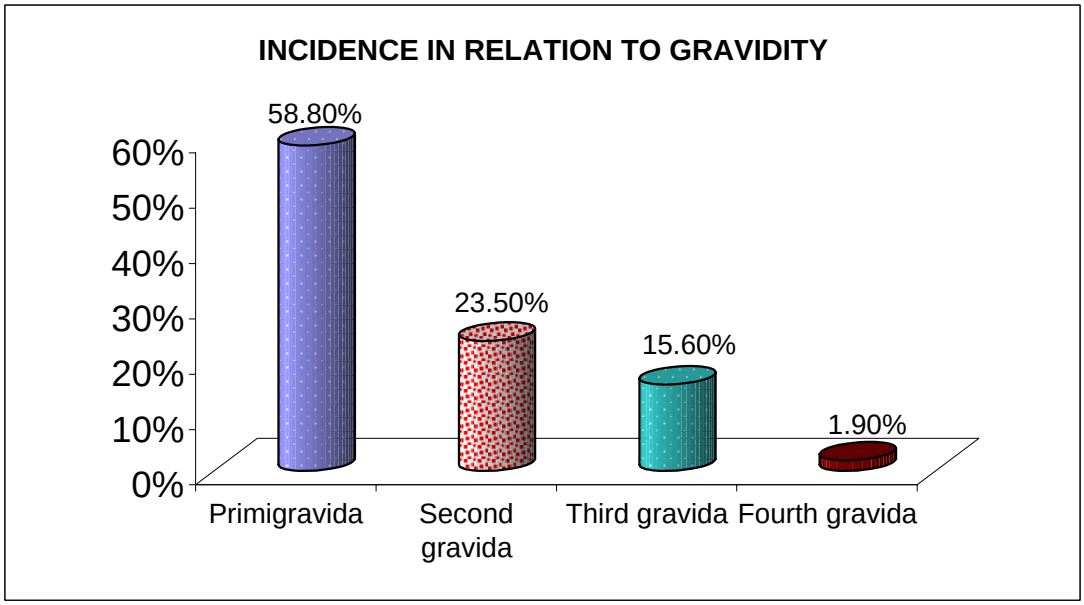

ANALYSIS OF ASSOCIATED COMPLICATIONS
30%
25%
20%
15%
10%
5%
0%
27.40%
7.80%
3.90%
1.90%
19.60%
3.90%
Anaemia
Pre-eclampsia
Encephalopathy
Hepatorenal failure
Postpartum haemorhage
Coagulation failure

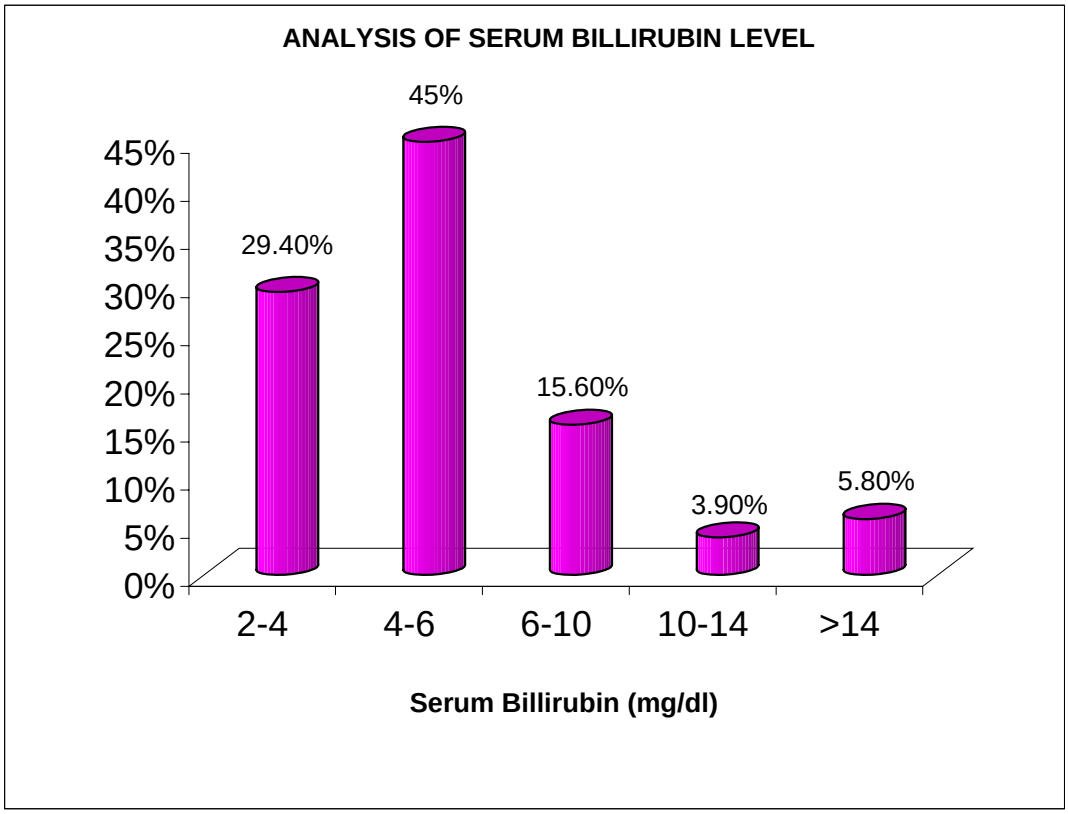

ANALYSIS OF BIRTHWEIGHT AND FETAL OUTCOME
30%
25%
20%
15%
10%
5%
0%
11.7%
11.7%
17.6%
9.8%
29.4%
0.0%
29.4%
1.9%
3.9%
1.9%
3.9%
0.0%
1-1.5
1.6-2
2.1-2.5
2.6-3
3.1-3.5
>3.5
Birth Weingt in Kg
Total Cases
Fetal Deaths

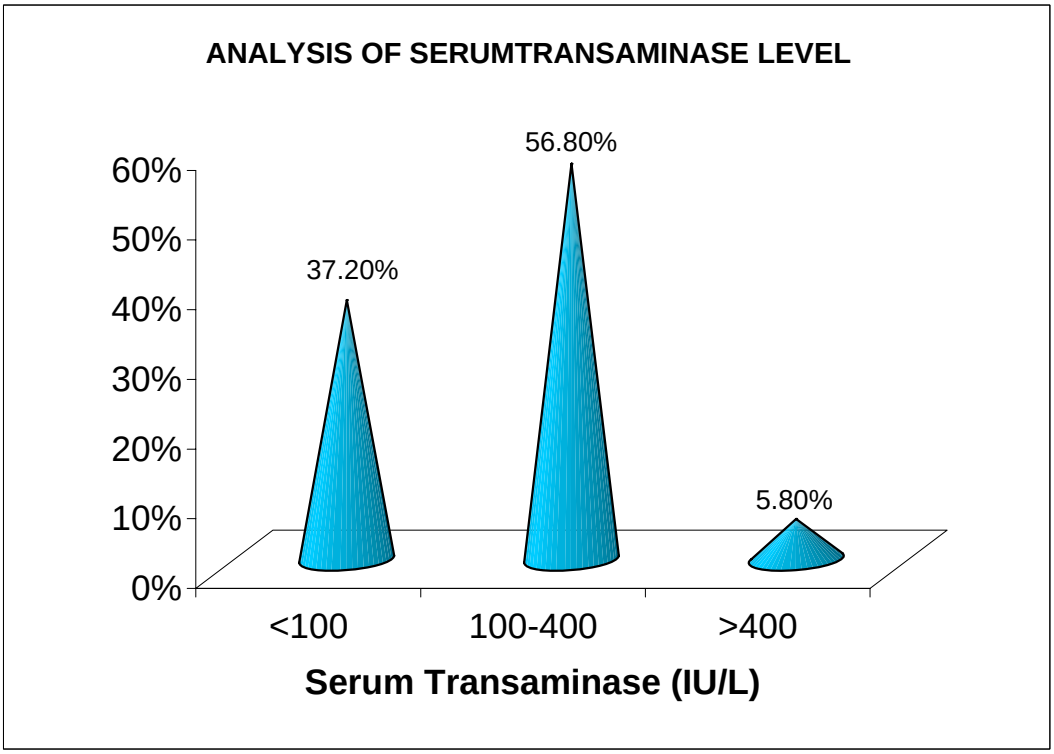
RELATION OF TYPE OF DISEASE TO MATERNAL MORTALITY

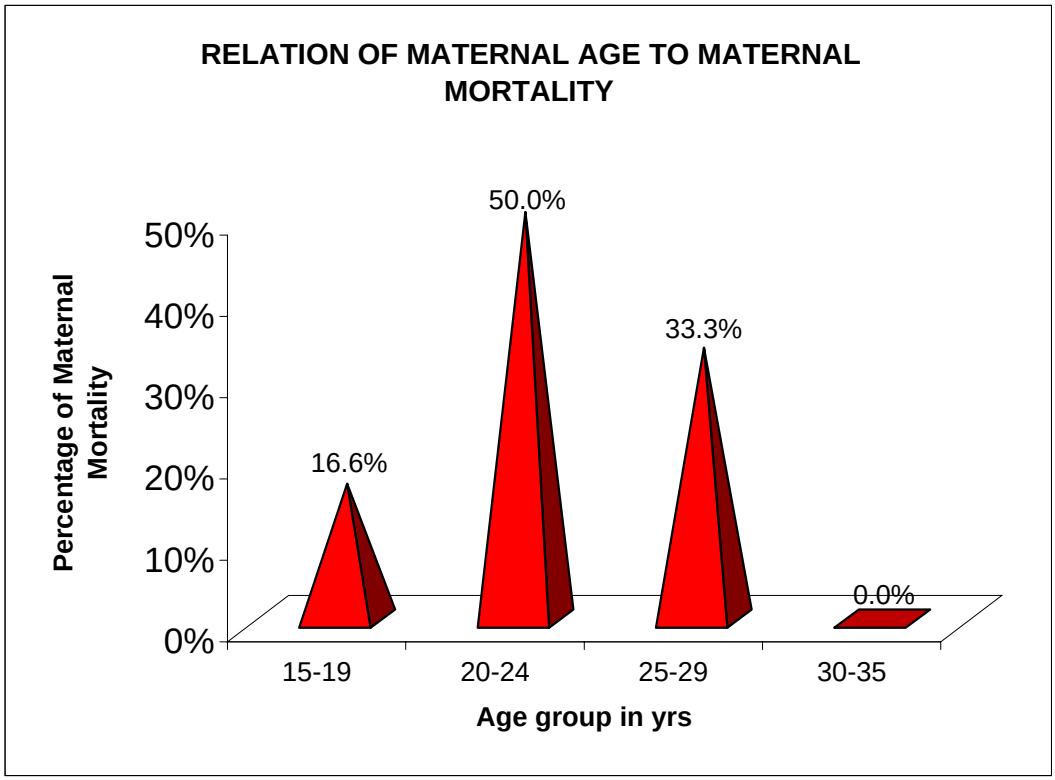


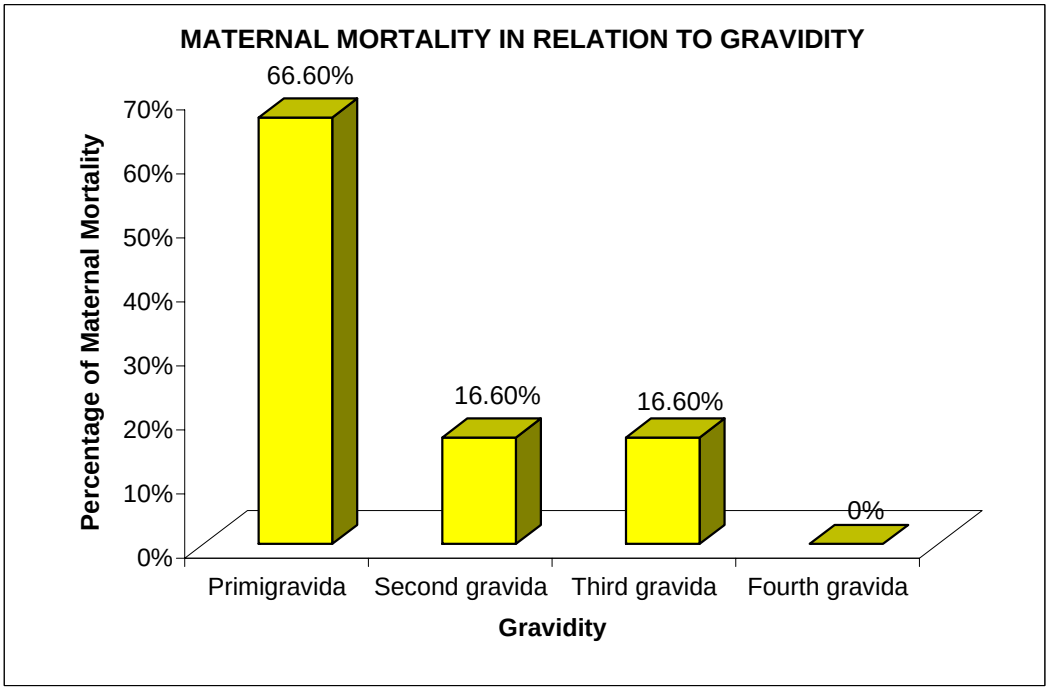

AGE DISTRIBUTION
60%
50%
40%
30%
20%
10%
0%
58.80%
29.40%
7.80%
3.90%
15-19 yrs
20-24yrs
25-29yrs
30-35yrs

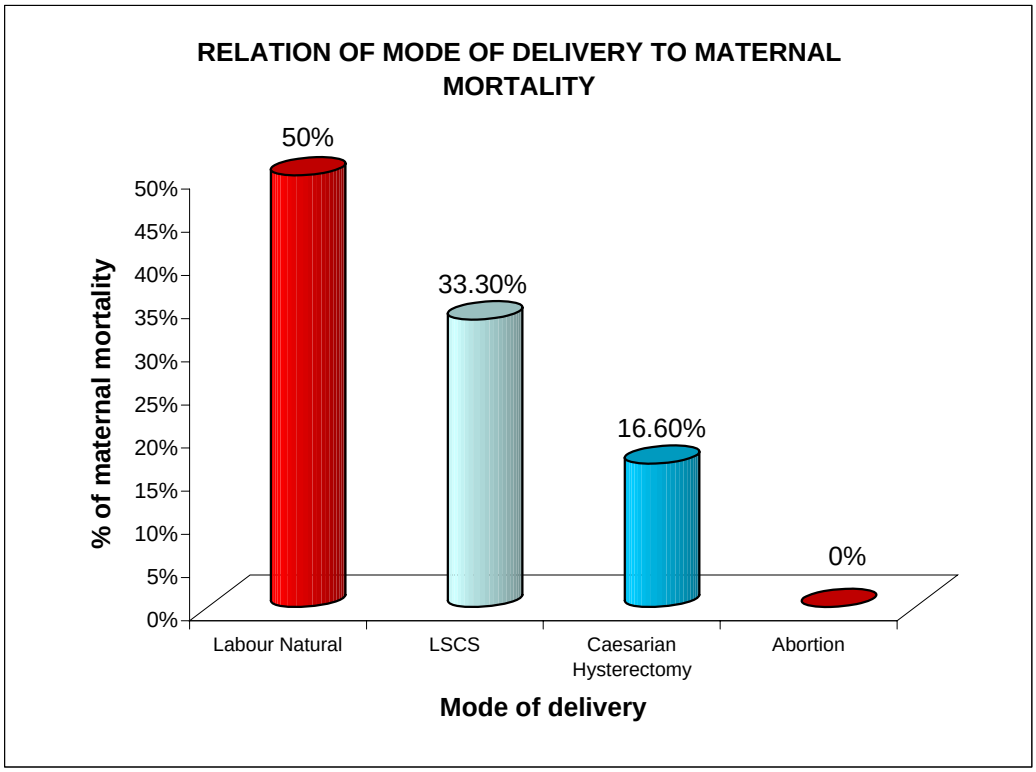

INCIDENCE IN RELATION TO GRAVIDITY
60%
50%
40%
30%
20%
10%
0%
58.80%
23.50%
15.60%
1.90%
Primigravida
Second gravida
Third gravida
Fourth gravida

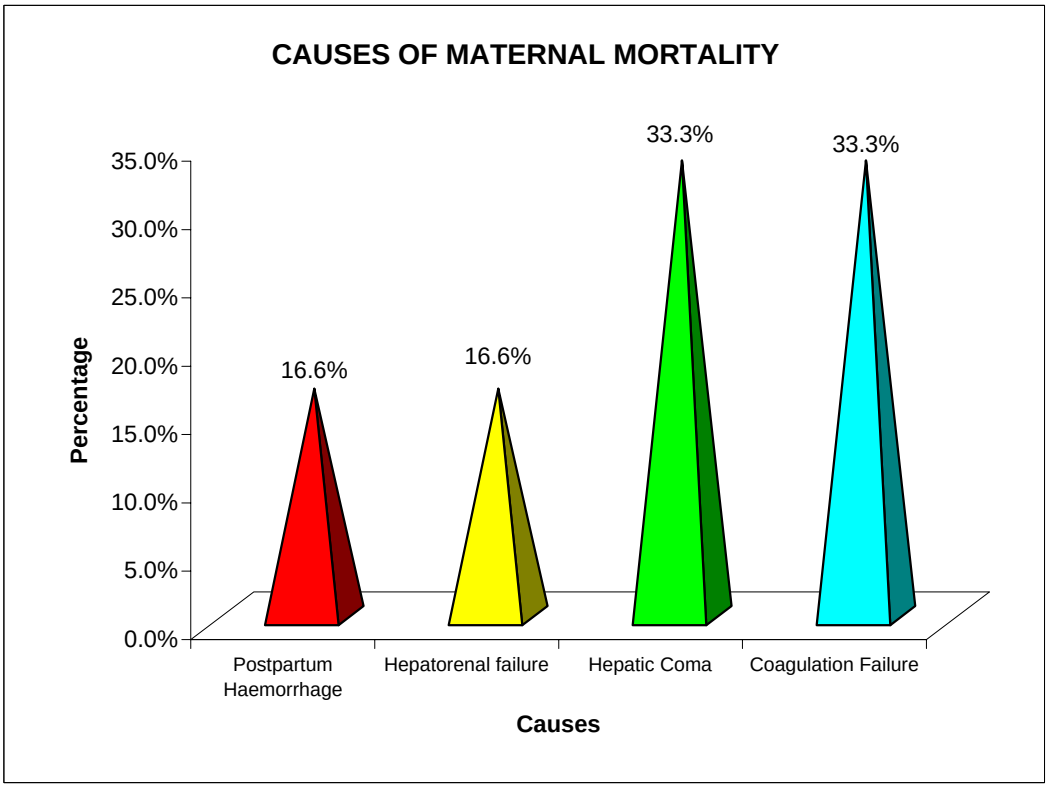

ANALYSIS OF SERUM BILLIRUBIN LEVEL
45%
29.40%
15.60%
3.90%
5.80%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
2-4
4-6
6-10
10-14
>14
Serum Billirubin (mg/dl)

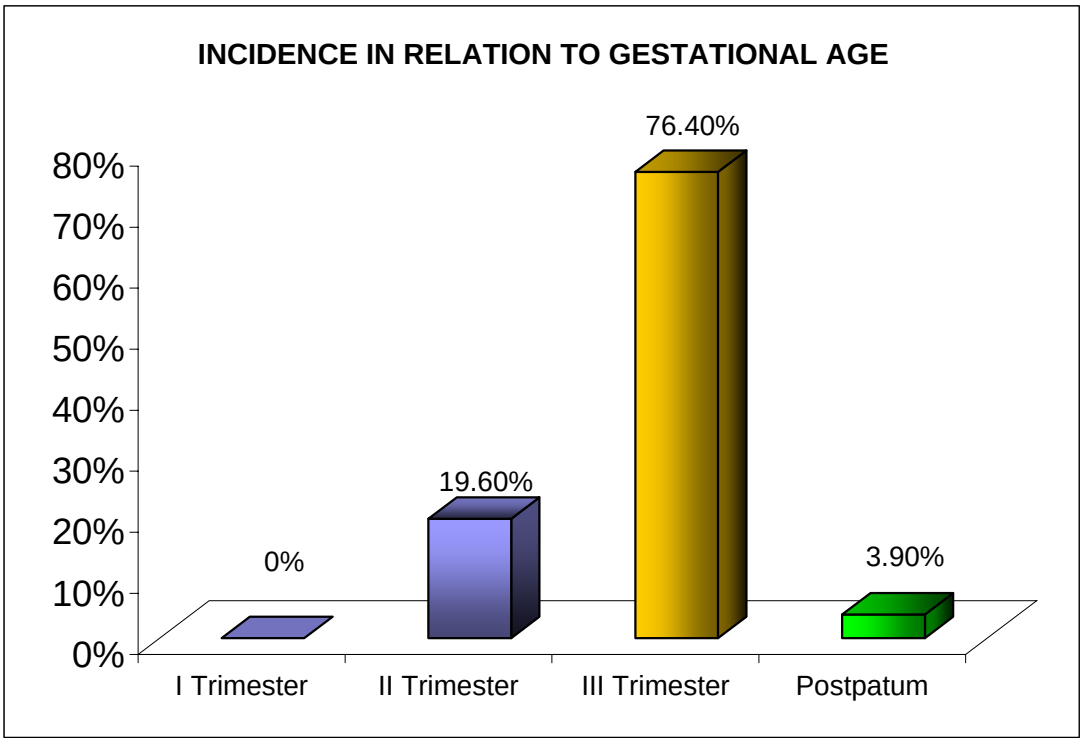

ANALYSIS OF SERUMTRANSAMINASE LEVEL
56.80%
37.20%
5.80%
60%
50%
40%
30%
20%
10%
0%
<100
100-400
>400
Serum Transaminase (IU/L)

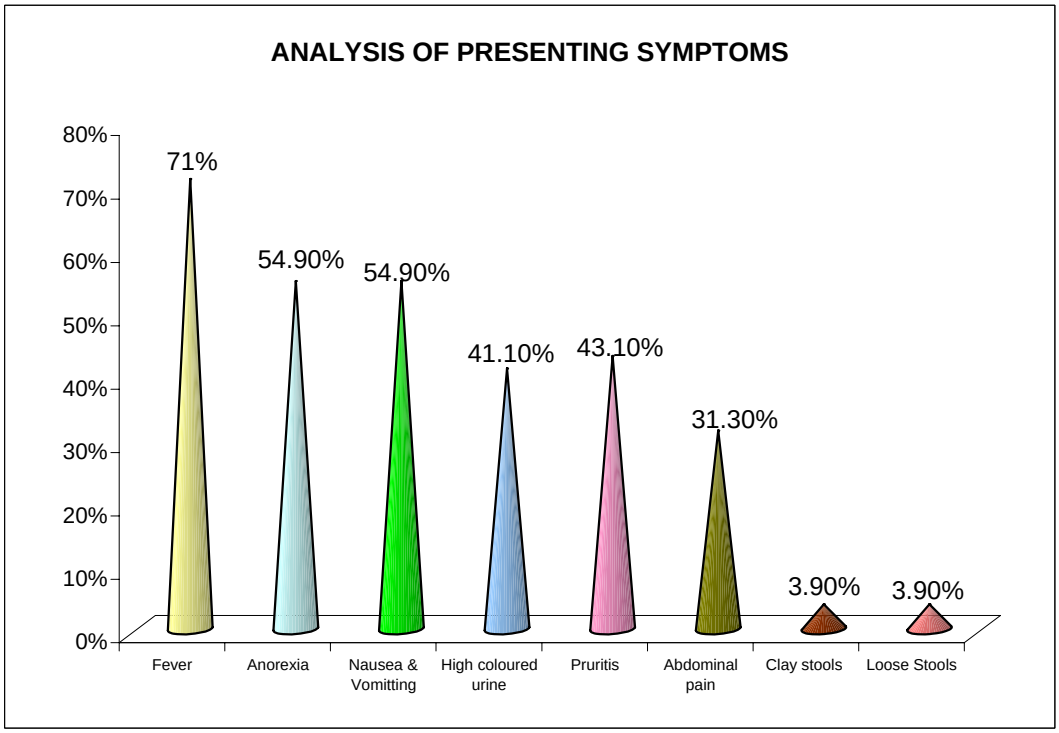

RELATION OF MATERNAL AGE TO MATERNAL MORTALITY
Percentage of Maternal Mortality
50%
40%
30%
20%
10%
0%
16.6%
50.0%
33.3%
0.0%
15-19
20-24
25-29
30-35
Age group in yrs

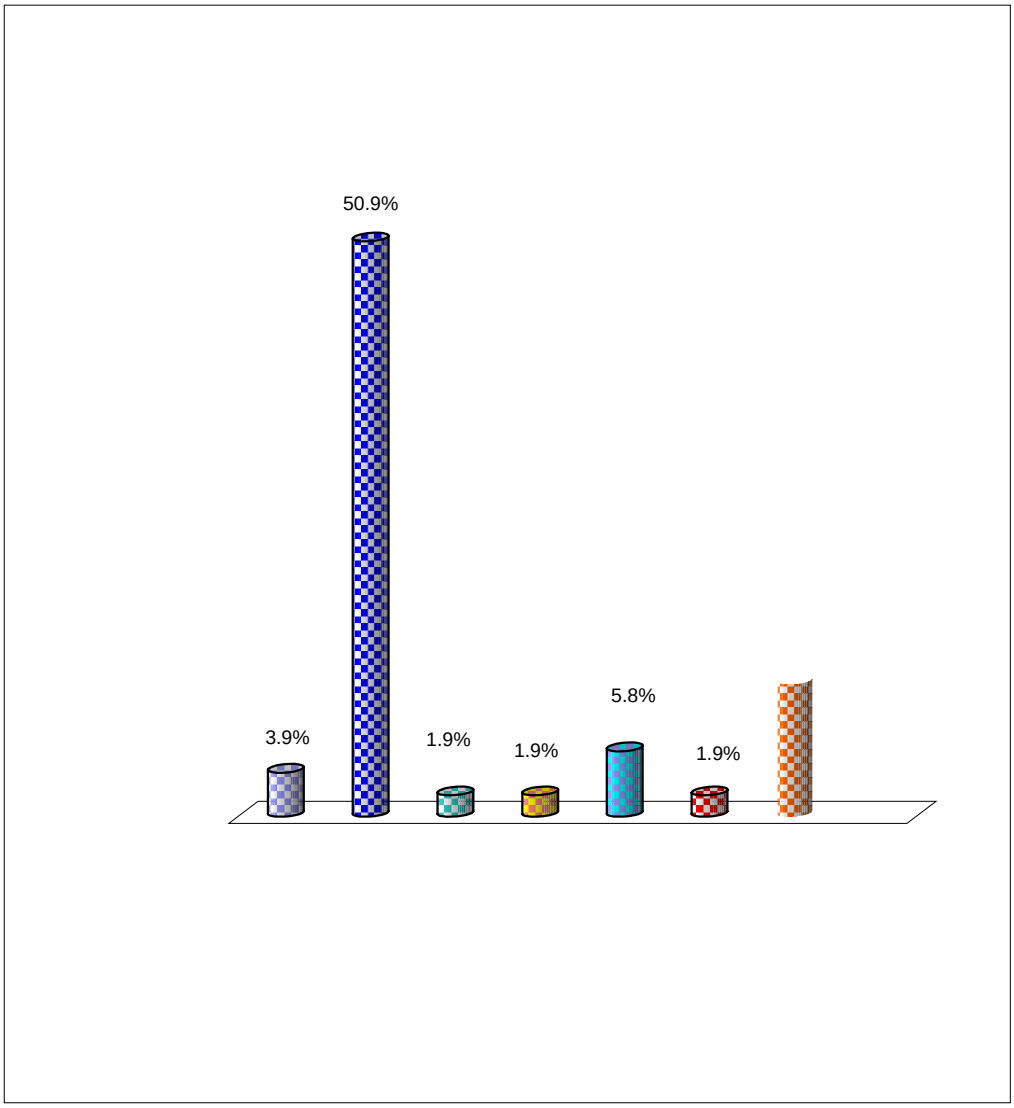

MATERNAL MORTALITY IN RELATION TO GRAVIDITY
Percentage of Maternal Mortality
70%
60%
50%
40%
30%
20%
10%
0%
66.60%
16.60%
16.60%
0%
Primigravida
Second gravida
Third gravida
Fourth gravida
Gravidity


RELATION OF MODE OF DELIVERY TO MATERNAL MORTALITY
% of maternal mortality
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
50%
33.30%
16.60%
0%
Labour Natural
LSCS
Caesarian Hysterectomy
Abortion
Mode of delivery


CAUSES OF MATERNAL MORTALITY
Percentage
35.0%
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
16.6%
16.6%
33.3%
33.3%
Postpartum Haemorrhage
Hepatorenal failure
Hepatic Coma
Coagulation Failure
Causes


INCIDENCE IN RELATION TO GESTATIONAL AGE
80%
70%
60%
50%
40%
30%
20%
10%
0%
0%
19.60%
76.40%
3.90%
I Trimester
II Trimester
III Trimester
Postpatum


ANALYSIS OF PRESENTING SYMPTOMS
80%
70%
60%
50%
40%
30%
20%
10%
0%
71%
54.90%
54.90%
41.10%
43.10%
31.30%
3.90%
3.90%
Fever
Anorexia
Nausea & Vomitting
High coloured urine
Pruritis
Abdominal pain
Clay stools
Loose Stools


50.9%
3.9%
1.9%
1.9%
5.8%
1.9%